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President's Page

KNOWLEDGE BRINGS STRENGTH

It has been said that a man's judgment is no better than the information he possesses. In the same sense this is substantially true of organizations. An organization is only as strong as its members, the information they possess, and the desire they have to serve. Our Texas Medical Association councils and committees have come to realize more and more that we are only as strong as our 119 county societies. If we do not have strong and forceful leaders in these local groups, we cannot hope to have a closely knit and forceful state organization.

At the request of the Board of Trustees and officers of the Association, the Committee on Public Relations lays stress in its 1953 program plans on strengthening the ties between the councils and committees and the county society presidents and secretaries.

The first step in this direction was taken at the second annual conference of county presidents and secretaries held in conjunction with the midwinter meeting of the Executive Council in Austin on January 17. The meeting, begun last year on an experimental basis, brings together one of the main units in our chain of operation.

The program was planned to cover three fields of work—administrative, socio-economic, and legislative. County society presidents must be kept advised of the developments in these three phases of medical activity in order to fulfill their obligations as administrative officers of the county societies. Further, they must keep posted on developments at all three levels—national, state, and local.

We were fortunate this year to have at the county officers' conference Leo Brown, director of the American Medical Association's Public

Relations Department, and Malcolm Lamborne, Jr., assistant editor of publications in the Washington office of the A. M. A. and formerly a reporter on the Washington *Evening Star,* to bring us up to date at the national level. N. C. Forrester, Philip R. Overton, and W. E. Syers ably presented the picture on the state level.

Present plans are to follow this state meeting with a series of district PR clinics at which local problems will be discussed and possible solutions worked out.

Still another effort to inform leaders in the county societies on problems of concern to them as individuals and as a profession—problems not directly scientific or medical in nature—will be made during the 1953 annual session, when several talks at a general meeting of the Association will be devoted to these problems. Experts in various phases of medical practice, medical organization, and medical relationships, some of them from other states, will present these talks. Although the meeting in January and the district clinics planned for later in the winter and spring cannot include all the membership of the Association, the program during the annual session is planned for every member able to be present. Final details of this program will be announced soon.

If our county societies are to become leaders in the community, we must revitalize them. It is only through informed and aggressive county society officers (and other members) that this can be accomplished. It is our hope that through the meetings mentioned herein can come the information. If society officers supply the enthusiasm and aggressiveness, our objective of better intra-professional relations will be obtained.

T. C. Terrell

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EDUCATION BY TELEPHONE

For the second time in Texas, members of the Texas Medical Association can take part in a series of telephoned medical discussions on the postgraduate level to bring them up to date on latest developments in medicine. The advantage of the telephone postgraduate broadcasts is that the average Texas doctor can benefit from the program merely by attending his county society meeting where arrangements have been made to "tune in" on the program.

The postgraduate telephone broadcast series conducted in 1952 by the Texas Medical Association attracted more than fifty county societies to hear one or all of the three programs in the series. With the technical details of production worked out and streamlined by the experience of the first series, the 1953 series of four programs is expected to be even more successful.

To cut down on production costs for the county societies tying in at their local telephone offices, the first two broadcasts in the series have been scheduled for February 17 and March 10, bringing them within a single fiscal month and thus necessitating only one bridging charge for both programs.

The subjects of the four programs for 1953 have been chosen by a special subcommittee of the Council on Medical Education and Hospitals to interest both general practitioners and specialists. The four panel discussions, which will originate in the Texas Medical Association building in Austin, are on the following topics: "Peptic Ulcer—Modern Concepts," "Poliomyelitis—Evaluation of Present Status," "Practical Management of More Frequent Obstetric Emergencies," and "Use and Abuse of Antibiotics."

With ideas in postgraduate education constantly changing, the methods of presentation and communication of knowledge likewise undergo change. Through standardized courses, television, and telephone broadcasts, to name a few of the modern techniques, the medical profession is keeping abreast of modern times and taking every opportunity to become better qualified to care for the sick.

NATIONAL MEDICAL LEGISLATION

With the Eighty-Third Congress of the United States now in session, legislation of interest to the medical profession is brewing and will be presented during the coming months. Several of these proposals are related to the medical and social security fields. Every physician, whether or not he has formed an opinion on these subjects, should acquaint himself with the facts and be conscious of the stand, if any, of the American Medical Association on these matters.

Doctor-Draft Extension

One of the most important issues this year in the eyes of the medical profession will be the doctor-draft law which expires July 1. The present issue will be the question of whether the law should be extended and if so, what changes should be made. Other questions to be considered in relation to this law are whether military doctors are spending too much time on military dependents and civilians who might obtain medical care privately, what effect the new Congress and cabinet will have on the medical officer requirements of the Armed Forces, and whether the Armed Forces have perfected a recruitment program to obtain doctors on a voluntary basis. Representatives of the A. M. A. are meeting with military officers and attempting to answer some of the questions. Until more information is known, the A. M. A.'s official position neither supports nor opposes extension of the law although the Association still adheres to the position which it took in supporting Public Law 779 when it was first presented that the calling of physicians in larger numbers than are needed to provide the necessary medical needs of the Armed Forces is objectionable.

Social Security Amendments

The question of waiver of Old Age and Survivor Insurance benefits for the permanently and totally disabled was a center of controversy last year. The A. M. A. did not oppose extension of the benefits but objected to provisions of the proposed law allowing the Federal Security Administrator to prescribe regulations for medical determination of total and permanent disability. A disability section written to expire June 30, 1953, was included in the law, but claimants were not to be allowed to apply for benefits until July 1. Therefore, the section becomes inoperative the day prior to its effectiveness. The new Congress is expected to hold public hearings on the waiver of premium issue soon.

Also of importance is the proposal for sixty days' annual free hospitalization for aged beneficiaries of the social security program. Eligible persons are estimated at 7,100,000 and the Federal Security Administration has set the cost at approximately $235 million a year.

Retirement for Self-Employed

For about a decade corporations have been allowed to establish pension funds out of tax-free money for their employees. In 1952 an effort was made to extend the privilege to the self-employed, including physicians, setting a limit of 10 per cent of income or $7,500 on the amount to be placed in such funds annually, the total not to exceed $150,000. Funds could not be withdrawn until death, retirement, or disability. No action was taken although public hearings were held by the House Ways and Means Committee. Actively supporting the plan were the A. M. A., the American Dental Association, the American Bar Association, and other groups representing the self-employed.

International Labor Organization

Action by the Senate is expected on the Bricker Resolution, providing for a constitutional amendment prohibiting United States participation in any international agreement affecting rights of American citizens or superseding the United States Constitution. The target of this action is the International Labor Organization, which proposed setting up minimum standards of social security including national health insurance through a convention which has the same force as a treaty. The affirmative vote of two-thirds of the Senate present would make

the proposal of the International Labor Organization effective. The official stand made by the A. M. A. at the Denver Clinical Session in December was to reaffirm support of the joint resolution prohibiting this action.

Executive Reorganization

After the Eighty-Second Congress rejected a bill to consolidate all health, medical, and hospital functions of the federal government into a Department of Health with cabinet status, the Senate Committee on Government Operations proposed instead a Federal Board of Hospitalization designed to bring about better integration of all federal medical activities and programs and to pass on plans for utilization of new and existing medical facilities. The A. M. A. supported the plan with several exceptions, including that there be a fair representation of physicians on the board and that the Veterans Administration not be shielded from recommendations and regulations of the board. It is likely that similar legislation will be introduced in the Eighty-Third Congress.

Local Public Health Units

New bills on local public health units are considered certain to be introduced in this Congress. The American Medical Association opposed a section of proposed legislation allowing almost unlimited activities for a local public health department partially supported by federal funds. The A. M. A. believed the bill's definition would stimulate local public health departments to expand into various fields of medical care. Federal Security Administration officials insisted on broad definition of public health services. An agreement was not reached and the bill was not reported out by the House Committee.

Emergency Maternity and Infant Care

New E. M. I. C. bills are expected to appear this year even though the Defense Department and Budget Bureau did not support bills presented last year. Federal support of a program for emergency maternity and infant care for dependents of military personnel was attempted by legislation in the last Congress. During the hearings many witnesses including A. M. A. representatives testified that no economic need exists at this time for such a program. The testimony was backed up by surveys made near military installations. Witnesses stressed that fathers are not being drafted and Armed Forces representatives testified that 57,118 deliveries were recorded in military hospitals in six months. A bill was reported out of the Health Subcommittee of the Senate Labor and Public Welfare Committee in the last days of the session but the bill was not brought to a vote in the Senate. New measures can be expected to be introduced this year.

Federal Aid to Medical Education

A bill for federal aid to medical education rejected by the last Senate was sent back to committee. The A. M. A. renews its stand that only if private and state support fails should medical schools seek aid from federal government. Also, the A. M. A. supports the theory that federal assistance if necessary should be in the form of one-time construction grants for building and facilities similar to the Hill-Burton hospital program. It is expected that bills on this subject will be brought up again.

These and other subjects of interest to physicians are expected to appear upon the political and legislative scene in 1953. Every doctor must be alert and watchful to make sure that he supports sound and favorable legislation and opposes any proposals which are not for the good of the American people and good medical care.

THE 1953 MARCH OF DIMES

Throughout 1952 the eyes of the nation were focused upon Texas, during the worst epidemic of poliomyelitis in the state's history. Of 51,600 new cases reported as of November 1 by the United States Public Health Service, 3,880 occurred in Texas. Also, Houston was the site of a large-scale experiment to determine the effi-

cacy of gamma globulin in allaying the disease or preventing it entirely.

Seven Texas cities received emergency equipment and volunteer workers to fight poliomyelitis last year. As in former years, the Lone Star State received help from the National Foundation for Infantile Paralysis in excess of her contributions to the annual March of Dimes. Despite the fact that the 1952 drive in Texas exceeded two million dollars, to rank third among states of the Union, deficits from the previous year plus the heavy financial toll of poliomyelitis in the state during 1952 means that the 1953 campaign, more than any previous drive, needs to succeed.

Texas doctors will want to urge families and friends to support the 1953 March of Dimes this month and to do all else within their power to see that poliomyelitis is defeated.

HEART DISEASE — A CHALLENGE AND AN ANSWER

Rheumatic heart disease, high blood pressure, and arteriosclerosis are the Big Three among the group of twenty or more diseases which affect the heart and blood vessels, believes Dr. Louis N. Katz, immediate past president of the American Heart Association. They account for 90 per cent of heart disease.

Research, physician education, public education—these are the tools used by national, state, and local heart associations to fight heart disease. While direct aid to patients is not possible, they benefit indirectly from the development of cardiac clinics, home nursing care, assistance in dietary problems, and social services.

Physician education proceeds along the lines of formal teaching, distribution of educational material, and the use of cardiac clinics for instruction in diagnosis and treatment.

A research foundation has been set up within the Texas association within the last year to see that all funds collected in heart campaigns within the state are put to full use; Texas is the first state to set up a foundation of this kind.

More has been learned about the human heart in the past twenty-five years, perhaps, than in all past history. New drugs, new methods of surgery bring hope to victims of heart disease whose cases would have been hopeless even a decade ago. To continue this progress will demand the active interest and financial support of the public. Physicians can be instrumental in gaining help for a cause they know is worth while, the Heart Association drive, which this year will be conducted during February.

NAMES OF COUNTY DELEGATES NEEDED

County medical societies have received requests to supply the Executive Secretary of the Texas Medical Association with the names of officers and committee members for 1953. These names are needed for a variety of reasons. Mail of all kinds must be directed to county society officeholders, and progress is expedited if the mail is correctly addressed.

A prompt report on delegates who will serve medical societies in the House of Delegates will assure that all eligible physicians are considered by the President when he selects reference committees. Dr. T. C. Terrell, who will appoint reference committees for the 1953 session, has expressed his desire to make the appointments well in advance of the meeting to permit each reference committee member to study the annual reports before he arrives in Houston, thus assuring a more leisurely and effective consideration of important business.

The effort to provide adequate representation of all parts of the state and of the various county medical societies in the important reference committees may well be stultified by failure of counties to report promptly the names of their delegates. Other projects also may be slowed down or well-nigh stopped because incomplete and outdated lists of county society officers are the only lists available.

ORIGINAL ARTICLES

Problems in Roentgen-Ray Diagnosis of Congenital Heart Conditions

E. W. SPACKMAN, M.D., D.Sc., Fort Worth, Texas

THE tremendous advances recently made in the knowledge of congenital heart conditions have completely changed our methods and criteria for diagnosis. Surgery has kept pace with the progress and many conditions considered hopeless a few years ago are now diagnosed accurately and the patient successfully operated upon. The criteria of the uncomplicated major types of congenital defects are well established. However, approximately 50 per cent of cases may not be definitely diagnosed by the correlation of clinical, physical, and laboratory procedures including the conventional roentgenograms and fluoroscopic evidence. Some workers[17] claim that accuracy in diagnosis without the use of the special procedures should be as high as from 80 to 85 per cent. In my experience, nearly 50 per cent of routine cases have shown features making it advisable to check the findings by one or more of the special techniques. These special procedures include tomography, kymography, angiocardiography, cardiac catheterization, and aortography.

TABLE 1.—*Distribution of Congenital Heart Conditions.*

Heart Condition	% of Cases
Acyanotic	
Interatrial septal defect (Lutembacher's)	20-25
Patent ductus arteriosus	10-15
Coarctation of aorta	8-10
Pulmonary stenosis	1-2
Cyanotic	
Tetralogy of Fallot	12-15
Eisenmenger's complex	5-8
Rare conditions	
Transposition of great vessels	1-2
Truncus arteriosus	1-2
Tricuspid stenosis	1-2

The latter three methods are not without a definite measure of risk and should never be advised without due consideration and clean-cut indications. Kymography and tomography give confirmatory evidence in certain specific conditions but are of accessory value only.

Table 1 shows the relative distribution of these cases in our service and agrees closely with those published by other workers.[10] It may be noted that defects of the interatrial septum and patent ductus arteriosis comprise the largest group within the acyanotic class, and that tetralogy of Fallot is the outstanding diagnosis in the cyanotic class. Mistaken diagnosis in those cases which are not absolutely typical may lead to unnecessary surgical procedures. For example, it is often advisable, in cases of apparently simple patent ductus arteriosus, to investigate further if there are any clinical or physical data suggesting possible complicating factors. As in other types of diagnostic radiology, the closest cooperation is an absolute requirement and the various workers should form a team which consult together constantly.

Errors of technique and interpretation creep into angiocardiography and confuse the results. The usual errors resulting from technique include too much dye, slow injection, spasm or resistance within the veins, unusual anatomic relationship of the veins, improper positioning, and cardiac arrest. Improper interpretation usually is found in false atrial defect (early filling of the right atrium with extension to the left), false subpulmonic stenosis (systole of the right ventricle produces dilatation of the pulmonary artery), false filling defect (upper half the right atrium may fill before the lower half), and false appearance of patent ductus arteriosus (aortic exaggeration produced by traction of the ligament and so forth).

It is especially important that the radiologist have these possibilities of error in mind. The selection of the special procedures and application to the individual case should be a function of the medical, surgical, and radiologic team. Each of the special studies contributes a different and specific set of data, and they are not selected at random but applied to the problem at hand. Angiocardiography, for example, is useful in studying the filling and emptying of chambers, size and configuration, and the great vessels. Catheterization will supply information on pressure readings and oxygen content within the vessels and chambers (right heart). Aortography provides visualization of the aorta and its tributaries. Kymography reveals the wave patterns of chambers and vessels. Tomography, which is rarely used, indicates masses or displacements. The choice of these special studies is suggested in table 2.

Read before the Section on Radiology, Texas Medical Association, Annual Session, Dallas, May 6, 1952.

Kymography may add valuable confirmatory evidence in certain problems (table 3).

It is the purpose of this paper, on the basis of the individual problem, to outline the various steps in establishing final diagnosis by simple clinical and conventional radiologic procedures, the value of the special procedures, and how they may be selected to bring out final diagnostic criteria.

TABLE 2.—*Choice of Special Studies in Congenital Heart Conditions.*

Study	Condition
Catheterization	Interatrial defects Intracardiac shunts
Aortography	Patent ductus arteriosus Coarctation of aorta
Angiocardiography	Tetralogy of Fallot Eisenmenger's complex Truncus arteriosus Transposition of great vessels
Kymography	Patent ductus arteriosus Coarctation of aorta Hilar vessels
Tomography	Lutembacher's defect Masses Displacements

PATENT DUCTUS ARTERIOSUS

The typical case of patent ductus arteriosus illustrated in table 4 usually requires merely the correlation of the clinical, physical, and plain film studies. It is recommended that films be made in the posteroanterior, lateral, and both oblique projections with the barium filled esophagus. Thurn recommends kymography[20] as valuable adjunctive evidence.

The clinician is often confronted with an infant showing a systolic murmur, possibly discovered accidentally. This in itself is sufficient reason for thorough study in an attempt to rule out other possibilities. The conventional roentgen-ray studies should always be done.

TABLE 3.—*Evidence of Congenital Heart Defects Offered by Kymography.*

Patent ductus arteriosus: accessory waves in pulmonary artery
Interauricular septal defect: increased pulsation of pulmonary artery and expansile hilar pulsations
Coarctation of aorta: increased wave amplitude above, decreased below; in the left oblique position with filled esophagus, shallow irregular waves at apex
Tetralogy of Fallot: flat waves with splint peaks at apex, mixed auricular pulsations on the right border, no expansile pulsations in the pulmonary artery (exception: poststenotic dilatations)
Eisenmenger's complex: same as in tetralogy of Fallot with increased expansile pulsations in the pulmonary area

After reasonable elimination of other conditions, especially rheumatic heart, pancreatic, and blood diseases, I believe that these cases may well be kept under observation and check-up studies made, including roentgen-ray surveys, at least yearly. In following the case, a typical picture may be recognized between the ages of 4 and 6 years, and if doubt still exists, special studies are often required. I prefer aortography as a means of identification and reserve catheterization for those few cases wherein there is confusion with possible interatrial septal defect. Angiocardiography is sometimes selected as the initial special procedure where the diagnosis appears to be further complicated. An excellent discussion of these problems is presented by Burchell.[2]

When the patient is first seen in the more advanced stages, the differential problems indicated in table 4 must be considered. In older patients, it is often important to establish the diagnosis promptly and rule out other factors without the delay which is allowable in very young infants. These cases fall into three classes: (1) those in which the diagnosis is certain from clinical and conventional roentgenograms, (2) those in which a single special procedure fully establishes the patent ductus, (3) those in which other complicating factors make the diagnosis extremely difficult by the most exhaustive studies. In a few cases of the latter group, surgical exploration has been the final answer, but I do not advise this approach unless all other means have been exhausted.

TABLE 4.—*Patent Ductus Arteriosus.*

Clinical Picture	Surgical Aspect
Underdeveloped Repeated pulmonary infection Dyspnea and palpation Machinery murmur (wide pulse pressure accompanying P_2)	Identification Estimation of size, type, and amount of blood shunt Compensating factor Strain on left heart Complicating conditions Bacterial endocarditis Repeated lung infections
Plain Roentgenogram	**Factors of Confusion**
Prominent pulmonary segment Enlarged left ventricle (left auricle) Prominent aortic arch Elevation of left pulmonary artery branch Hilar congestion and dance (10%)	Infancy (defect not typical until third to sixth year) Without cardiac enlargement Systolic or atypical murmurs "False" evagination Compensation for other congenital conditions
Special Studies	**Differentiation**
Aortography: evagination, immediate filling of pulmonary vessels Angiocardiography: evagination, enlarged left ventricle, late filling of pulmonary branches Catheterization: increased oxygen content of the pulmonary artery past the level of the ductus Kymography: abnormal wave peak after usual pulmonary artery impulse	Interatrial defect (Lutembacher's) Infantile coarctation Rheumatic heart disease Venous hums and anemias Rare conditions Abnormal venous insertion Infantile aortic or pulmonary regurgitation Rupture sinus of Valsalva

INTERATRIAL SEPTAL DEFECT

Interatrial septal defect is one of the most common congenital cardiac anomalies. Clinically, it may strongly simulate conditions which may be helped by surgical intervention. It is therefore necessary to identify this entity or exclude it. It is to be hoped that on the basis of work now being done, patients with inter-

atrial septal defect will become candidates for corrective surgery, but to date no operation has been perfected.

The clinician is confronted with symptoms which are subject to wide variation (table 5). This is especially true at birth and in infancy. In this group, there may be no murmur or there may be a low systolic murmur. According to Taussig[19] there is nothing to differentiate this murmur from the systolic murmur associated with patent ductus arteriosus or ventricular septal defect. Cosby and Griffith[3] expressed the belief that the physical signs in adults may vary so widely that the presumptive diagnosis of interatrial septal defect depends upon radiologic evidence. The

TABLE 5.—*Interatrial Septal Defect.*

Clinical Picture	Special Studies
No cyanosis (transient cyanosis in a young infant)	Catheterization: increased carbon dioxide content of right auricle
Palpitation, fibrillation, congestive failure	Angiocardiography: revisualization of right heart at late stage
Systolic murmur (snapping P_2)	Kymography: increased pulsation of pulmonary artery and hilar vessels
Low blood pressure	
Right axis deviation in electrocardiogram	**Differentiation**
Plain Roentgenogram	Patent ductus arteriosus
Enlarged heart (right auricle and ventricle)	Lutembacher's complex
Prominent pulmonary artery	Rheumatic heart disease
Hypoplastic aorta	Persistent atrioventricularis
Left atrial enlargement (Lutembacher's)	Vessel anomalies
	Dilated pulmonary artery
	Pulmonary veins draining into the right auricle

characteristic roentgen features are indicated in table 5. Circulation time studies, if positive, strongly support the diagnosis. A negative result, however, is probably not of great significance, although the value of this test has not yet been clinically proved. Electrocardiographic findings are helpful in the uncomplicated case.

Angiocardiography studies are of limited value. Steinberg, Grishman, and Sussman[18] were able to demonstrate the defect only once in 10 attempts. My impression is that direct visualization of the defect is extremely unusual, but simultaneous visualization of the 4 cardiac chambers has been a good sign of interatrial septal defect if the dye is injected rapidly and in the correct amount.

The most reliable diagnostic aid is cardiac catheterization (table 5). Anatomic proof of the presence of interatrial septal defect is obtained by passing the catheter tip into the left atrium. Its position may be checked by fluoroscopic observation and film studies. It is also generally accepted that difference of 2 volumes per 100 or more between superior vena caval blood and right atrial blood is usually found when a significant opening between the atria exists.

COARCTATION OF AORTA

The clinical and physical features of the average typical case of coarctation of the aorta of the adult type are conclusive and the clinician has no difficulty in arriving at a correct diagnosis by correlating the clinical, physical, and plain film findings of the chest (table 6). The typical erosion of the under surface of the ribs does not often appear until the patient is between 12 and 14 years of age. Plain roentgenograms, particularly in the left anterior oblique projection, on occasion may give an indication of narrowing in the course of the upper aorta but can never be relied upon. Cardiac catheterization also has no place in this diagnosis except in rare instances where associated intercardiac shunts are suspected.

Kymography is sometimes useful by determining the wave pattern transmitted to the barium filled esophagus by the aorta. In those cases in which the height of the transmitted waves shows an abrupt and decided decrease, the level of the coarctation may be recognized. Recently, electrokymography has been used as a means of locating the position of the lesion.

TABLE 6.—*Coarctation of Aorta.*

Anatomic Picture	Plain Roentgenogram
Infantile	Small aortic knob
Proximal to ductus	Slight cardiac enlargement (left ventricle)
Open ductus	Rib fenestration
No collaterals	
Associated with other intracardiac shunts	**Special Studies**
Adult	Aortography: narrowing dilated proximal arch and neck vessels
At or distal to ductus	Angiocardiography: left ventricle dilated, dilated ascending aorta and neck vessels
Ductus closed	Visualization of position of narrowing: sometimes poor visualization of distal segment
Extensive collaterals	Kymography: difference in wave pattern above and below segment
Clinical Picture	**Surgical Aspects**
Infant	Size, location, extent
Blood pressure difference (early stage—with cyanosis of lower extremities)	Snub distal to subclavian artery (room for end to end anastomosis)
Harsh systolic murmur	Proximal dilatation (friability)
Right ventricular enlargement (right axis deviation to normal)	Extent of collateral circulation
Early decompensation (exertional dyspnea)	
No satisfactory surgical intervention	
Adult	
Hypertension (upper)	
Hypotension (lower)	
Basal systolic murmur (diastolic murmur means relative aortic insufficiency)	
Left ventricular hypertrophy (left axis deviation)	
Pulsations in collaterals	
Corrective surgery possible	

Angiocardiography, in young children, gives definite and reliable results indicating the location and extent of the narrowed segment and showing the adequacy of the collateral circulation. In some cases, however, the injected dye is too dilute on reaching the area under investigation to permit certainty of details. Aortography, therefore, is the method of choice

and gives exact data in regard to the type and extent of the narrowed portion, guiding the surgeon in planning the approach and measures for relief (table 6).

Special investigations in the infantile types of coarctation are extremely important in those cases in which there is a chance of operative intervention. The clinical and physical findings are subject to greater degree of confusion than in the adult type. Patients seen very early present a rather typical picture, but as the child develops, and especially since the infantile type is often associated with other intracardiac shunt, the typical findings become overshadowed. A right to left shunt causes the child to become cyanotic, and the right axis deviation, which is often present in the early stage, almost always disappears as the child develops.

Importance of early recognition and operation, when possible, in these cases is stressed by Olney and Stevens.[13]

I therefore believe that aortography offers the greatest value in cases of adult coarctation. Where diagnosis is in question at a moderately early age, angiocardiography and sometimes catheterization have been the initial methods of choice. In the infantile type, angiocardiography has always been chosen as the initial special procedure.

TETRALOGY OF FALLOT

When the clinician is confronted with the cyanotic child, recognition of the condition and the probability of operative intervention is of special importance. It is especially essential to rule out the more serious conditions (truncus arteriosus and so forth) which are not amenable to surgical relief. Tetralogy of Fallot is the most common of the cyanotic group.

The clinical diagnosis is simple when the history, physical signs, and radiologic findings follow the classical description (table 7). The "textbook" case presents a history of early persistent cyanosis and exertional and paroxysmal dyspnea with squatting for relief. Physical examination reveals a stunted cyanotic person exhibiting a weak pulse with a narrow pulse pressure. The heart action is forceful with basalar systolic murmur and trill. The pulmonic sound is poor. Laboratory study reveals polycythemia with deviation of the electrical axis to the right. The roentgen organ shows "dry lungs" with the typical cor en sabot silhouette (table 7).

In the case of an infant less than 12 months of age, the diagnosis of tetralogy of Fallot may be difficult or even impossible. The cyanosis may be questionable and the auscultation and roentgen findings may be indecisive. After two years, there is definite cyanosis, clubbing, history of dyspnea, and other unusual findings and the roentgenographic changes become definite. According to prevalent opinion, surgery is preferably deferred until after infancy.

The differential diagnosis may include Eisenmenger's complex, transposition of the great vessels, gross septal defect, pulmonary artery venous fistula, pulmonary stenosis with septal defect, and common truncus (table 7). In problems of this nature, the special procedures of angiocardiography and cardiac catheterization are invaluable. This is particularly true when the cyanosis is mild. Poststenotic dilatation of the pulmonary artery may be present. These special procedures identify the lesion and establish the possibility of successful surgery.

TABLE 7.—*Tetralogy of Fallot.*

Clinical Picture	Surgical Aspect
Early persistent cyanosis	Identification
Paroxysmal dyspnea	Size of main pulmonary artery divisions
Squatting	Location and character of pulmonary stenosis
Basal systolic murmur	
Polycythemia	
Right axis deviation in electrocardiogram	Differentiation
Plain Roentgenogram	Eisenmenger's complex
Boot shaped heart (concave upper left)	Pulmonary stenosis with septal defect
Right ventricle enlargement	Common truncus
Aorta displaced anteriorly	Large ventricular septal defect and right to left shunt
Diminished hilar and linear markings	

Angiocardiography demonstrates the over-riding aorta with simultaneous opacification of the right ventricle, aorta, and pulmonary artery. The amount of dye entering the pulmonic circuit and returning to the left atrium may be estimated and is helpful in obtaining preoperative information as to the degree of stenosis, and to what degree the circulation may be expected to increase by anastomotic procedure. The best surgical approach is also established by angiocardiography. Approximately 20 per cent of tetralogy of Fallot patients present a dextro-aorta descending on the right. Cardiac catheterization may directly demonstrate the overriding aorta by passing from the right ventricle to the ascending aorta. Oxygen saturation and pressure studies elucidate the presence of pulmonary stenosis and overriding aorta but I have depended primarily on angiocardiography in these problems.

EISENMENGER'S COMPLEX

Eisenmenger's complex is similar to tetralogy of Fallot in that there is dextro-position of the aorta and right interventricular septal defect. It differs from tetralogy in that there is no pulmonary stenosis and usually not as marked right ventricular hypertrophy.

Eisenmenger's complex is relatively infrequently seen. Characteristically, patients with Eisenmenger's complex develop persistent cyanosis late in childhood or early adult life. The intensity of cyanosis may vary from day to day, but the intensity increases gradually over a period of years. There may be hemoptysis, clubbing, hoarseness, or brassy cough. A hoarse systolic basal murmur and thrill are characteristic. The heart is usually slightly enlarged and may exhibit a normal pulmonary artery or considerable prominence. The electrocardiogram is usually little or no help. Determination of arterial oxygenation levels and circulation times are useful.

A difficult problem of differential diagnosis arises in certain cases which present clinical features of Eisenmenger's complex but with changes on an entirely different anatomic basis (table 8). Such conditions are tetralogy of Fallot, with mild pulmonary stenosis and poststenotic dilatation, high interventricular septal defect with considerable pulmonary hypertension and reversal of shunt, and pulmonary hypertension with reversal of blood flow through a patent foramen ovale. Angiocardiography reveals simultaneous opacification of the right ventricle, aorta, and pulmonary artery in these conditions.

TABLE 8.—*Eisenmenger's Complex.*

Clinical Picture	Differentiation
Cyanosis and polycythemia somewhat late	Tetralogy of Fallot
Other findings similar to tetralogy of Fallot	Lutembacher's defect
	Rheumatic heart disease
	Cor pulmonale
Plain Roentgenogram	
Enlarged heart (right ventricle)	
Prominent pulmonary artery (segment and vessels)	

Cardiac catheterization may demonstrate dextroposition of the aorta, absence of pulmonary stenosis with identical pressures in the pulmonary artery, aorta, and right ventricle. If these facts can be demonstrated, the cardiac catheterization is of great diagnostic aid, but I have depended largely on the angiocardiogram as the initial method of choice when this condition is suspected.

RARE CYANOTIC HEART MALFORMATIONS

Certain congenital cardiac malformations are always associated with cyanosis and usually terminate in early death. Among these malformations are absent aortic arch, aortic atresia, complete transposition of the great vessels, tricuspid atresia, and truncus arteriosis. These conditions are rare and only tricuspid atresia, complete transposition of the great vessels, and truncus arteriosis will be considered.

Tricuspid Atresia

Tricuspid atresia is often associated with other heart and great vessel anomalies. If development has been orderly, there must be at least an associated right to left interatrial shunt and a patent ductus arteriosus with flow from the oarta to the pulmonary artery. These shunts are essential to life. There are invariably associated nonfunctional right ventricle and pulmonary valve.

The clinical diagnosis is based on severe, persistent cyanosis present at birth and the demonstration of left deviation of the electrical cardiac axis. The universally poor prognosis and the diagnostic reliability of left axis deviation on the electrocardiogram usually makes special studies unnecessary. Angiocardiography shows early visualization of the left heart and aorta, no right ventricle, and poor or nonvisualization of the pulmonary artery. If a special study has not been done and the patient survives past the age of 2 years, the possibility of tricuspid atresia with ventricular septal defect and with an extreme stenosis of the infundibulum of the right ventricle should be considered (table 9).

TABLE 9.—*Rare Cyanotic Conditions.*

Clinical Picture	Plain Roentgenogram
TRICUSPID ATRESIA	
Intense cyanosis	Large left ventricle (small right ventricle)
Left axis deviation in electrocardiogram	Concave left border and clear pulmonary window
Special Studies	Dilated superior vena cava
Angiocardiography	
TRANSPOSITION OF GREAT VESSELS	
Clinical Picture	**Plain Roentgenogram**
Cyanosis (less in lower extremities)	General cardiac enlargement
Special Studies	Narrow great vessel shadows in antero-posterior position
Angiocardiography	Wide great vessel shadows in left anterior oblique position
TRUNCUS ARTERIOSUS	
Clinical Picture	**Plain Roentgenogram**
Cyanosis (intense)	Cardiac enlargement
Increased pulse pressure	Prominent "aortic knob"
Special Studies	Shelving of right ventricle (left anterior oblique position)
Angiocardiography	Diminished hilar shadows

Transposition of Great Vessels

Complete transposition of the great vessels results in two independent circulations. The left heart delivers blood to the lungs and receives the pulmonary blood. The right heart receives the somatic venous return and delivers blood to the aorta. To be compatible with life, there must of necessity be intracardiac and/or extracardiac shunts. The greater the volume of these shunts the more favorable is the prognosis. At best, this is always poor.

Clinically, the patient presents intense cyanosis with

early progressive cardiac enlargement. The distribution of cyanosis may be diagnostic. The lower extremities may be less cyanotic than the upper. Thrills and murmurs are of no diagnostic aid. Routine roentgen-ray examination of the infant is difficult, but there is marked enlargement of the ventricles, and there may be rhythmic change in the size of the right atrium independent of heart beat. The electrocardiogram usually shows right axis deviation. The differential diagnosis includes truncus arteriosus, pure pulmonary stenosis (advanced grade), and anomalous drainage of the pulmonary veins into the right auricle and extreme tetralogy of Fallot (table 9).

Truncus Arteriosus

A truncus arteriosus is a single large vessel that receives all the output of both ventricles. In some cases, this vessel may divide so that there is arterial flow to the lungs and body or the blood may be supplied to the lungs by bronchial arteries. In the latter case, cyanosis is severe. If a pulmonary artery arises from the truncus, cyanosis may be minimal. Associated anomalies are common. These facts cause considerable variance in the clinical picture. The roentgen-ray examination shows great cardiac enlargement with absence of the pulmonary artery segment and marked prominence of the aortic knob and left ventricle. The left anterior oblique views show marked shelving of the right ventricle. As the patient becomes older, the cardiac contour approximates that of tetralogy of Fallot. The electrocardiogram is usually of no specific help. The differential diagnosis includes a nonfunctioning right ventricle and extreme dextro-position of the aorta combined with pulmonary atresia. The angiocardiogram shows a single large vessel arising from the heart and is diagnostic (table 9).

TABLE 10.—*Choice of Methods to Study Congenital Heart Defects.*

PATENT DUCTUS ARTERIOSUS	TETRALOGY OF FALLOT
Usual	Usual
Clinical, physical, and laboratory data	Clinical, physical, and laboratory data
Plain roentgen-ray studies	Plain roentgen-ray studies
Exceptional	Angiocardiography
Aortography	Exceptional
Angiocardiography	Catheterization
Kymography	Kymography
INTERAURICULAR DEFECT	**EISENMENGER'S COMPLEX**
Usual	Usual
Clinical, physical, and laboratory data	Clinical, physical, and laboratory data
Plain roentgen-ray studies	Plain roentgen-ray studies
Catheterization	Angiocardiography
Exceptional	Exceptional
Angiocardiography	Catheterization
Kymography	Kymography
COARCTATION OF AORTA	**RARE CYANOTIC CONDITIONS**
Usual	Clinical, physical, and laboratory data
Clinical, physical, and laboratory data	Plain roentgen-ray studies
Plain roentgen-ray studies	Angiocardiography
Aortography	
Exceptional	
Angiocardiography	

CONCLUSIONS

Roentgen-ray diagnosis in congenital heart conditions is arrived at by summation and analysis of all possible material. The possibilities are then estimated objectively and correlated with evidence from the clinical, physical, laboratory, and plain roentgenogram findings. Special radiologic studies are advised whenever indicated but with due regard to the danger involved. There can be no rigid routine, but table 10 indicates the general manner of procedure. Each case is judged independently on its own merit and the value of teamwork is especially stressed.

I wish to extend sincere thanks to Dr. D. R. Bernhardt for his assistance in compiling the manuscript, to Dr. Clive Johnson for his suggestions and review of the material, and to Mrs. Frank Gladden for the secretarial work.

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ABSTRACT OF DISCUSSION

DR. J. E. MILLER, Dallas: Dr. Spackman has written exhaustively on the problems of diagnosis of congenital heart conditions. As might be expected, he has not confined his discussion to the roentgen-ray diagnosis of congenital heart conditions. It is obvious to me that as one learns a great deal about any subject it is impossible to confine oneself to a single phase of this subject. The radiologist, therefore, cannot talk about congenital heart disease from the roentgen point of view without discussing some of the clinical problems.

Many clinicians have considered it their prerogative to obtain all the data about a certain condition, then put it all together and make the diagnosis. There are still some radiologists who would be satisfied to interpret roentgenograms objectively. If the patient's best interests are of primary importance to the physician, the radiologist will have much clinical data at his finger tips when interpreting films, or it should be available to him in personal consultation with the clinician. This is especially true in congenital heart disease. I have several hundred cases of congenital heart disease easily available at my finger tips. I frequently amuse myself by asking various cardiologists and other internists to give me their impression of what might be present from an objective inspection of adequate roentgen-ray films. As might be expected they do rather poorly with this objective interpretation. I also frequently ask for an objective interpretation of these films by radiologists and they do but little better. It is surprising how frequently they can arrive at the correct diagnosis after asking two or three questions or being given one or two points of the clinical data. To illustrate better the point that I want to get over, I would like to relate an actual occurrence.

It is my good fortune to be associated in Dallas with Dr. Gladys Fashena. She occupies a position of preeminence in Dallas insofar as congenital heart disease is concerned, similar to that occupied by Helen Taussig in Baltimore. On one occasion we were having a rather heated discussion about the diagnosis in a particular patient. We obviously were disagreeing. In desperation I asked Dr. Fashena what were her clinical reasons for the diagnosis that she was willing to make. In enumerating the various points she said, "And the roentgen-ray examination shows this." I immediately objected and said, "Dr. Fashena I am interested in the clinical reasons for your diagnosis, not the roentgen manifestations." Her immediate reply was to the effect that she considered the roentgen-ray examination one of the most important parts of the clinical examination in the study of congenital heart disease. If more physicians would adopt this attitude and realize that they are on the same team rather than give lip service to this attitude, the patient certainly would benefit.

It is obvious Dr. Spackman has gone a long way in increasing his ability to diagnose congenital heart disease.

Angiocardiography fails on many occasions to give an exact anatomic diagnosis. If one is aware of the limitations of this examination, he is not surprised. Despite this fact, however, it still has much value in patients in whom the exact anatomic diagnosis could not be known. Angiocardiography frequently will give information concerning the physiologic abnormality in cases where anatomy is not known. This is important because the physiology of the heart is much more important than its anatomy. To illustrate: I have been unable on several occasions to say positively that a patient had tricuspid atresia rather than tetralogy of Fallot. Since the problem of physiology is much the same, I was certainly able to indicate that pulmonary stenosis was present and that there was a vascular ischemia to the lungs. This was important information to the clinician and naturally made it apparent that the patient was a suitable candidate for a shunting procedure.

There are several minor points in this paper in which my experience is somewhat at variance with that of the author, but this is to be expected.

Use of New Drug for Rheumatoid Arthritis

Promising results in the administration of a new drug, phenylbutazone, in treatment of rheumatoid arthritis and other similar musculoskeletal disorders was reported in two articles in the November 15 issue of *The Journal of the American Medical Association*.

One article, reporting results of treating 147 persons suffering from rheumatoid arthritis and rheumatoid spondylitis, states that the drug produces striking subjective improvement in a high percentage of rheumatoid spondylitis and is less effective in peripheral rheumatoid arthritis. The writers stress that the drug can cause serious side effects and use of the drug should be limited until more studies have been made. The article also reports that it has not been determined whether the drug is truly antirheumatic or only analgesic in its action.

In the second article describing a study of 200 trials of phenylbutazone and of a combination of phenylbutazone and aminopyrine, the authors express preference for phenylbutazone alone as the possible pharmacologic hazard of aminopyrine is eliminated. Fifty-one toxic reactions were seen in this series, and although none were serious, it was necessary or advisable to cease therapy in 22 of the patients. Most reactions subsided when use of the drug was discontinued.

Both articles reported the drug's effectiveness became apparent within a few days. When appreciable benefits were obtained, they rapidly reached a maximum and improvement remained level throughout the period of treatment. Most patients noted a return of symptoms within three days after cessation of therapy.

Tuberculosis is notorious for the difficulty of detecting it in its early stages. By the time it is discovered, the disease usually has been transmitted to others. In view of this fact, no method of treatment will solve the TB problem in short order, although improved methods should help hasten the far-off day of eradication of tuberculosis. A good vaccine, superior to BCG—the one most widely used at present—would have greater potentialities for rapid control of tuberculosis than any method of treatment. There is no simple solution to the tuberculosis problem. The disease, because of its infectiousness, its present prevalence, its symptomless onset, its long duration, its tendency to recur even though once arrested, is one of the most complex and costly public health problems in the United States.—James E. Perkins, M. D., Annual Report of the National Tuberculosis Association, April 1, 1951 to March 31, 1952.

Tuberculosis among diabetics is a special, serious, and integral part of the whole problem of eradicating tuberculosis. The importance of early isolation of the patient with positive sputum should be stressed, as well as prompt hospitalization and institution of appropriate therapy. Reeducating the patient so that he will practice good living habits will do much to prevent future relapses.—David A. Cooper, M. D., and Katharine Boucot, M. D., Am. J. Nursing, Nov., 1952.

Pulmonary Embolism; Its Prevention and Treatment

R. D. HOLT, M.D., Meridian, Texas

NOWHERE in the field of medical and surgical practice is found anything so shocking as the sudden unexpected death from pulmonary embolism. The sudden blow frequently occurs practically without warning. Great strides in medicine such as improved surgical techniques, advances in anesthesia, discovery of the antibiotic drugs, and improved maternal and infant care have been made. These advances have materially lowered the mortality rate in each instance. However, the mortality of pulmonary embolism is still far too high.

In a series of 172,888 postoperative cases studied at the Mayo Clinic, the incidence of pulmonary embolism was 0.52 per cent and of fatal embolism 0.20 per cent. According to these figures, one of every 200 patients who submit to surgery will have a pulmonary embolism and one of every 500 patients will die from an embolism.[1] Dr. Irving S. Wright has stated that it is the most common pathologic process producing the terminal fatal episode in the lives of patients over 50 years of age.[7]

PREVENTION

In the prevention and treatment of pulmonary embolism, it is most important to prevent venous thrombosis. If thrombosis in the veins of the lower extremities can be avoided, the possibility of fatal pulmonary embolism is all but eliminated. Phlebothrombosis occurs almost silently without signs of inflammation. Thrombophlebitis, as the name indicates, presents all of the signs of acute inflammation. It is the almost silent phlebothrombosis which is responsible for nearly all of the cases of pulmonary embolism.

Factors important in the prevention of venous thrombosis are (1) early ambulation—walking, not sitting; (2) avoidance of constrictive abdominal bandages and binders; (3) avoidance of Fowler's position in bed; (4) the use of leg exercises in all bed patients, especially bicycle riding movements and dorsiflexion of the foot and ankle (particularly important in patients over 40); (5) gentle handling of tissues at surgery, and (6) judicious use of the anticoagulant drugs.

The use of alpha-tocopherol and calcium has been found to act as an antithrombin in preventing thrombosis by Ochsner and others in New Orleans.[5] Several new anticoagulants have been developed. Two of these are Tromexan and BL-5 (4-hydroxycoumarin anticoagulant no. 63, Abbott) which appear to be effective in prolonging the prothrombin time and less dangerous than Dicumarol. A heparin-like substance, Paritol, is also under study.[6]

Even if venous thrombosis occurs after the precautions listed are observed, much can be done if the condition is recognized and treated early before fatal embolism can occur. It is easy to overlook the early signs of venous thrombosis which occur when treatment could be most effective. There may be no definite physical or subjective signs but nearly always there is a slight rise in pulse rate and temperature. The finding of a positive Homans' sign,[3] that is, pain in the calf of the leg on forceful dorsiflexion of the foot, is almost diagnostic. Slight swelling about the ankle or calf of the leg and dilatation of superficial veins of the foot also are suggestive of venous thrombosis. Any complaint of leg pain, no matter how slight, must be considered significant.

If the physician will be diligent in watching for any of these early signs, many cases will be discovered early with correspondingly improved results.

TREATMENT

When pulmonary embolism does occur in spite of careful treatment, the first attack is frequently not fatal. Therefore, it is of utmost importance to diagnose the attack correctly. It is easy to consider it some sort of heart pain, pleurisy, or even hysteria. Too often valuable time in starting treatment is lost by not suspecting pulmonary embolism in every attack of chest pain or dyspnea in a bed patient.

The active treatment of pulmonary embolism and venous thrombosis after either or both have occurred is essentially the same. Should it be medical with the anticoagulant drugs (heparin and Dicumarol) or surgical by superficial femoral ligation or vena caval ligation? A recent analysis by Linton of thrombo-embolic disease at the Massachusetts General Hospital reveals that of 799 patients treated with Dicumarol, there were 2 deaths related, in part at least, to the drug. Of 1,041 prophylactic superficial femoral vein ligations, 5 resulted in death.[4] Neither route, therefore, is entirely reliable. I believe that either method instituted early will materially improve results. I lean toward medical treatment with venous thrombosis and in apparently small pulmonary emboli. On the other hand, repeated embolic episodes and those of apparently severe degree have been treated with ligation with satisfactory results.

PRESENT SERIES

An analysis of the 10 cases of pulmonary embolism which I have seen during the past ten years in a small

Read before the Section on General Practice, Texas Medical Association, Annual Session, Dallas, May 6, 1952.

general hospital will be presented. Most of the cases were in the medical and traumatic groups rather than the surgical or obstetric groups. This was also true in a ten year analysis at the Massachusetts General Hospital, where the incidence on the surgical service was only 0.24 per cent and on the medical service 0.6 per cent.[2] During this period, in our hospital, there were 9,120 general admissions to the hospital with 10 cases of pulmonary embolism, an incidence of a little more than .1 per cent, or approximately 1 in 1,000 that were diagnosed. Of these, 2,278 were surgical with 2 cases of pulmonary embolism, an incidence of less than .1 per cent; 1,127 were obstetric with 1 case of pulmonary embolism, an incidence of less than .1 per cent; the balance of admissions (including many pediatric patients) were combined medical and traumatic cases with 7 cases of pulmonary embolism or an incidence of more than .1 per cent. The criteria of diagnosis of these cases were primarily a sudden attack of chest pain after eight to twenty days in bed followed a day or so later by coughing up bloody sputum and usually some roentgenographic evidence. Finding evidences of venous thrombosis in one or both lower extremities added confirmation to the diagnosis.

TABLE 1.—*Summary of 10 Cases of Pulmonary Embolism.*

Case	Year	Age (Yr.)	Diagnosis	Day Emboli Occurred	No. of Emboli	Results
1. Mrs. S. R.	1944	68	Peritonitis, ruptured appendix	13	2	Recovery
2. Mrs. W. A.	1945	62	Hysterectomy, cystocele and rectocele repair	8	1	Fatal
3. Mrs. A. A.	1945	29	Postpartum and tube ligation	17	1	Recovery
4. Mrs. T. C.	1946	49	Hysterectomy, cystocele repair	15	1	Recovery
5. Mr. G. P.	1947	56	Fracture femur	21	3	Recovery
6. Mr. E. G.	1947	51	Mumps orchitis	10	1	Recovery
7. Mrs. C. M.	1949	58	Contusion of calf of leg	15		
			Thyroidectomy	2	2	Recovery
8. Mr. J. H.	1949	35	Severe electric burn	12	1	Recovery
9. Mr. A. J.	1950	54	Fractures of tibia and ninth dorsal vertebra	15	1	Recovery
10. Mr. J. L.	1951	67	Coronary thrombosis	44	1	Recovery

In this group of 10 cases there was 1 fatality. This is the only case with any doubt about the diagnosis; an autopsy was not obtained. However, the chart did show a slight increase in pulse rate and possibly a little edema of the right ankle before sudden death on the eighth postoperative day.

The following case histories are rather typical.

CASE 1 (Obstetric).—Mrs. A. M., a 29 year old white woman, in 1945 delivered her fourth child without difficulty and had a fallopian tube ligation the second postpartum day. The patient remained in bed ten days. Seventeen days after the birth of her child, while washing diapers she was seized by a sudden severe substernal pain, great respiratory distress, and a feeling that she was about to die. The patient gave a history of acute thrombophlebitis in the right lower extremity after the birth of her third child two years previous.

Examination showed marked cyanosis, great respiratory distress, and a feeling of impending death. Homans' sign was positive in the right leg. The patient began coughing up bloody-colored sputum the day following admission. Roentgenograms of the chest supported the diagnosis of a large pulmonary embolism. Treatment was conservative, that is, oxygen for cyanosis and dyspnea, sedatives for pain and rest. Anticoagulants were not used. Recovery was complete in four weeks.

CASE 2 (Medical).—E. G., a white man aged 51, had been in bed at home ten days with mumps orchitis and was admitted to the hospital in 1947 with pleurisy-like pain in the right side of the chest. Roentgenograms suggested an atypical pneumonia. Bloody sputum appearing the following day suggested pulmonary embolism. Further examination showed edema of the right ankle and a positive Homans' sign. Treatment was conservative with anticoagulants. Recovery was complete in four weeks.

CASE 3 (Surgical).—Mrs. W. A., a white woman aged 62, died suddenly in 1945 eight days after undergoing hysterectomy and cystocele repair for prolapse of the uterus although she apparently had been doing well. The patient was ready to return home, was ambulatory, and at the time of her death had no leg complaints or other signs suggestive of phlebothrombosis or thrombophlebitis. An autopsy was not secured. Death was assumed without confirmation to be due to pulmonary embolism.

CASE 4 (Traumatic).—J. H., a 35 year old white man, was admitted to the hospital in 1949 unconscious as a result of electric shock from contact with a high tension wire. He suffered third degree burns of the palms of the hands and soles of the feet. Twelve days after admittance he developed a sudden severe pain in the right side of his chest, shortness of breath, and considerable anxiety. The patient had a positive Homans' sign, and considerable swelling occurred in his left foot, leg, and thigh. Immediate ligation of the superficial femoral vein was performed with removal of an 11 inch thrombus the size of a lead pencil. When the vein was found, it was opened directly over the thrombus. Suction was applied with a glass drinking tube. Recovery was complete in eight weeks. No sequela was noted after two years. A brief analysis of the cases is reported in table 1.

SUMMARY AND CONCLUSIONS

The importance of the prevention and treatment of pulmonary embolism is presented with suggestions for early diagnosis of both venous thrombosis and pulmonary embolism. An analysis of 10 cases is presented.

It is not possible to draw any conclusions from this small group of cases. However, a few important facts stand out:

1. Pulmonary embolism is not primarily a surgical problem; it is even more common in medical and traumatic patients.

2. Early diagnosis of venous thrombosis is essential if treatment is to be effective in preventing pulmonary embolism.

3. The mortality of pulmonary embolism should not be high in well managed cases.

4. Pulmonary embolism must be suspected in every case of chest pain or dyspnea in a bed patient.

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ABSTRACT OF DISCUSSION

DR. J. ROSS SHIPP, Waco: In a considerable number of patients general practitioners see surgical, medical, obstetric, and injured patients who are all candidates for pulmonary embolism. One should again emphasize the manner in the prevention of venous thrombosis as described by Dr. Holt. A characteristic and unexplained feature of pulmonary infarction is the development of jaundice in an appreciable number of patients, particularly cardiacs in congestive failure. In such persons the appearance of jaundice, unless otherwise explained, should raise the suspicion of pulmonary infarction.

Care should be used in the aged in ligation of femoral and saphenous veins simultaneously, as their collateral circulation is not up to par. Embolism which would be sublethal in a young person might prove fatal in an older person. Active treatment as mentioned before is ligation, oxygen, sedation, and the use of anticoagulants with Dicumarol and heparin being most commonly used. Again, care should be used in old persons because of possible cerebral hemorrhage. Anticoagulant therapy may reduce mortality after pulmonary embolism or infarction from 16 to 18 per cent to less than 1 per cent. Tolerance to Dicumarol is variable and not related to weight, age, or sex. Doses may range from 175 mg. to 800 mg. weekly. The dose which will prevent thrombosis and yet not induce hemorrhage is often difficult to determine. Prothrombin time may be dangerously prolonged by slight overdosage, alcoholic excess, or aspirin.

BENIGN PERICARDITIS

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BENIGN pericarditis has been recognized for many years, but not until the last decade have more complete descriptions and studies of it been made. As the name indicates, it does not cause death, but in the more severe cases fear is often held for the life of the patient and it is often confused with more serious affections of the heart. This disease frequently is mistaken for some other serious cardiac disease with which the patient is labeled with the result that his future life becomes unfavorably altered. It has been confused with and mistakenly diagnosed as myocardial infarction, and the need for accurate differentiation in this instance cannot be too strongly stressed.

In 1854 Hodges[7] reported in the *Boston Medical and Surgical Journal* what apparently was the first recognition of benign pericarditis. He had referred to this disease as "idiopathic pericarditis," calling attention to the rarity of such cases not associated with rheumatism or tuberculosis, and observed that such cases occurred from exposure to cold or when no exciting cause could be found. The frequency of an associated infection of the upper part of the respiratory tract has been observed all through the years.

Another of the earliest rather accurate descriptions of this benign disease and the recognition of its association with an infection in the upper respiratory tract was reported by Morison[11] just forty-five years ago. He described this type of condition as differentiated from those of much more serious consequence, such as that associated with rheumatic fever and the infectious ones due to tuberculosis and the pneumococci.

It is noted that little if any space is devoted to this disease in the best known textbooks of our day, even the most recent ones. It is often thought of as a rare disease. Although it is not common, it is not too rare. In 292 cases of acute pericarditis observed at the Presbyterian Hospital[8] over a period of fifteen years, 7.5 per cent were classified as being of undetermined cause; the cases were carefully chosen, excluding any in which there was a possible known etiologic factor. Over a period of about twenty years there were 50 cases classified from the records of the Massachusetts General Hospital and the private services of some of the staff members.[3] In a report of 46 cases occurring in the Army during the last war reported by May and Boyer in 1945,[13] 15 cases were designated as pericar-

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ditis of undetermined origin, a much higher incidence than that observed in other series.

ETIOLOGY

As mentioned previously and as implied in the names given to this disease — benign, nonspecific, idiopathic, and pericarditis of undetermined cause — the etiologic problem remains unsolved. The four most commonly considered causes are as follows:

Virus Infection.—Observers who believe that a virus is the most likely cause point to the fact that the disease is commonly associated with or shortly follows a nonspecific infection of the upper part of the respiratory tract or pneumonia. The fluid removed from the affected pericardium has repeatedly shown absence of bacterial flora, but a virus has not been isolated. In many observations there has been generally a lack of leukocytosis. Barnes and Burchell[1] reported in 1942 that the greatest percentage of their cases of acute pericarditis of unknown etiology had been preceded by an infection of the upper part of the respiratory tract. It has been observed that pericarditis classified in this category has been found to occur concomitantly with a virus pneumonia. In 1945 Dathe and Nathan[12] reported 6 cases of acute pericarditis which had occurred with or followed acute infections of the upper respiratory tract and were manifested by effusion and electrocardiographic changes compatible with acute pericarditis. Studies of the effusion fluid in 5 cases were negative for bacteria. In 1950 Feder, Hoffman, and Sugar[5] called attention to the frequency of associated acute infections of the upper respiratory tract and acute primary pericarditis, and also the frequency of recurrent attacks. In 23 of the 27 cases reported in 1950 by Levy and Patterson[8] an infection of the upper respiratory tract or pneumonia of the primary atypical variety had preceded or accompanied the pericarditis. They observed, as others have, the frequency of recurrences. Carmichael and others[3] reported in 1951 that 29 of their 50 patients had had an antecedent respiratory infection of one of the common varieties. One case[4] is reported occurring without antecedent infection of the upper part of the respiratory tract in a man ten days after he visited a patient ill with acute benign pericarditis.

Aureomycin, which has proven value in virus infections, was used by Taubenhaus and Brams[16] in 3 cases of acute nonspecific pericarditis which they thought definitely responded well to the usual doses of the drug.

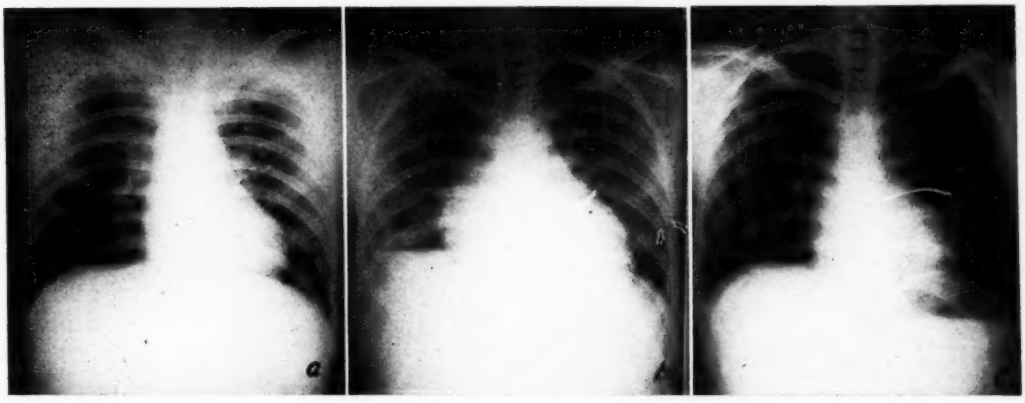

FIG. 1a. Chest roentgenogram of patient 10 made in 1948, when he was hospitalized for a fracture of the tibia.
b. Roentgenogram of patient 10 made October 30, 1950, soon after his admission to the hospital for benign pericarditis and at about the time a negative pericardial tap was done.
c. Roentgenogram of patient 10 taken eight days after the one in *b*. The patient at this time was asymptomatic.

The proximity of the hilar and tracheal lymph nodes to the pericardium is thought to have some relationship to the association of acute infections of the upper portion of the respiratory tract and pneumonia, and also involvement of the pericardium concomitantly or shortly following the respiratory tract infection with direct regional spread of the causative organism or process.

Tuberculous Infection of Pericardium.—Even though repeated and thorough studies of the cases classified as benign pericarditis have failed to show the tubercular organism as the causative organism, many believe that it still may be implicated. Cultures both on media and in laboratory animals have been negative. Acute pericarditis occurring along with the Ghon complex has been observed and by some has been considered to be benign. Wallgren in 1947 re-

ported 2 such cases. Breresford reported in 1949 a similar case, and after a follow-up of more than two years, the patient remained well and the electrocardiogram was normal. Benign pericarditis is often associated with a pleural effusion and has been considered similar to the frequent idiopathic pleurisy and effusion found in young people which has long been considered to be due to tuberculosis. Often the Mantoux reaction is positive but organisms are never isolated and proof is not absolute. Those cases due to tuberculosis usually have a prolonged unfavorable course with fever and signs of cardiac failure; they infrequently include a complaint of chest pain, which is the most common thing in the benign type. In the former, effusion tends to recur repeatedly after taps, whereas in the latter it usually does not reform after it is once aspirated.

Response to Unknown Toxins.—Because of the unknown cause of this pericarditis and the often implicated "toxins" in other such diseases of unknown origin, it has also been mentioned as a cause in this

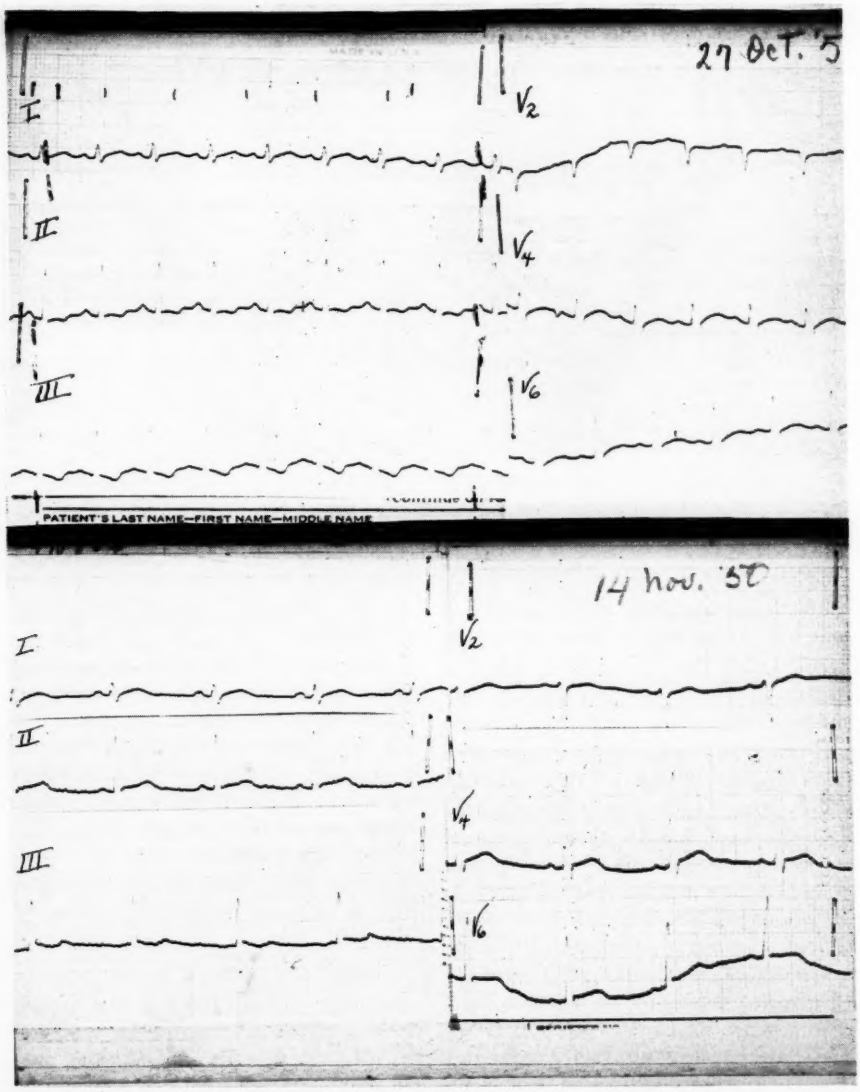

FIG. 2. Electrocardiogram of patient 10 taken on admission October 27, 1950, showing inversion of T waves in limb leads II and III and in V_2, and also some flattening in V_4 and V_6. A tracing done November 14, 1950, shows a return to normal of the T waves.

instance. Up to this time there has been no definite proof that any such "toxins" are present.

Hypersensitive Response of the Pericardium.—Hypersensitivity as a possible cause for benign pericarditis has many proponents. The condition often definitely follows an acute disease of the upper respiratory tract and may be due to hypersensitivity reaction localized to the pericardium from sensitivity to some bacteria or virus in the respiratory tract. Such a response parallels most of the present concepts for the manifestations in rheumatic fever and acute glomerulonephritis. McKinlay[10] reported a case of nonspecific pericarditis occurring in association with serum sickness. In 1949 Zivitz and Oshlag[20] reported a case of pericarditis in an allergic subject who developed a sterile effusion which contained 80 per cent eosinophils. There had been no response to antibiotic therapy and the course of the disease was somewhat prolonged, but when administration of an antihistamine was started, recovery followed rather shortly. An eosinophilia has been observed in other cases.

CLINICAL FEATURES, SIGNS, AND SYMPTOMS

From the literature, 185 cases of benign pericarditis that could be used were found and our 12 cases give a total of 197 cases reviewed. Tabulation of these varied widely, and the exact breakdown desired is not possible. As best as could be determined from the review, benign pericarditis occurs more frequently in whites than Negroes in a ratio of about 5 to 1, and more commonly in males than females in about a 6 to 1 ratio. The average age in the 197 cases is 34.3 years; ages ranged from 3 to 68 years.

As mentioned previously, the presence of concurrent or antecedent respiratory infection is a common finding. The onset is usually acute but may be gradual. All authors mention chest pain as the most constant symptom. Porter, Clark, and Porter[14] found it present in 100 per cent of their cases and observed that it was the essential symptom requiring relief. The pain is described as "sharp and knifelike" in most cases and is usually relatively intermittent. The pain is fairly well localized substernally or to the precor-

TABLE 1.—*Summary of Signs and Symptoms in 12 Cases of Benign Pericarditis.*

Case	Age (Yr.)	Hosp. Days	Up. Resp. Inf.	Friction Rub On Adm.	Dur. (Days)	Highest Hosp. Fever (F.)	Highest WBC Count	Cardiac Sil. F larged on Adm.	Ret. to Normal	Sed. Rate Range (mm.)	Electrocardiogram Changes	Ret. Norm.	Chest Pain Rel. by Sit. up	Agg. by Mov. & Cough	Remarks
1	21	32	Yes	Yes	12	103.4	17,400 Eosinophilia	Yes	Yes	20 5	Elevated ST; inverted T None	Yes	Yes	Yes	Pleural effusion
2	42	21	Yes	Yes	3	99.8	9,250	No		18 3			No	Yes	
3	35	35	Yes	No		101	16,750	No		32 19	Elevated ST; inverted T	Yes	Yes	Yes	Similar episode 45 days ago
4	25	14	No	No		98.6	6,700	No		14	Elevated ST; inverted T	Yes	Yes	No	Pneumothorax for TB
5	51	30	No	Yes	6	101	11,050	Yes	Yes	38 1	Inverted T	Yes	Resting on Back	Yes	
6	33	42	No	Yes	16	101.6	7,400	No		?	Elevated ST; inverted T	Yes	None	Known	Diabetic acidosis; supravent. tachy., pulse 214
7	53	30	No	Yes	1	102.4	14,350	No		40 32	Elevated ST; peaked T	Yes	?	Yes	
8	35	101	Yes	No		102	11,700	Yes	Yes	40 30	Inverted T	Yes	Yes	Yes	Pleural effusion; aureomycin neg.
9	24	9	Yes	No		101.4	9,100	No		30 10	Elevated ST; low T	Yes	Yes	Yes	
10	21	25	Yes	Yes	7	99.2	1? 600 Eosinophilia	Yes	Yes	40 18	Inverted T	Yes	Yes	Yes	Aureomycin neg; peri. tap neg.
11	52	55	Yes	Yes	1	104.4	16,100	Yes	Yes	42 25	Inverted, flat to diphasic T	Improved	Yes	Yes	Pleural effusion; aureomycin neg.; 2 peri. tap neg.; supra-vent. tachy
12	36	14	Yes	Yes	1	101	11,700	No		35 12	Elevated ST; flat to diphasic T	Yes		Yes	Similar episode 3½ mo. ago
Average	36	34	8 yes	8 yes	6	101.3	8 elev.	7 norm.	All	12 elev.	11 abnorm.	10	7 yes	10 yes	
Range	21-53	9-101			1-16	98.6-104.4	6,700-17,400			14-42					3 Pleural effusion 3 Aureomycin, all neg. 2 Peri.tap, no fluid 2 Recurrences

dium. One of the chief characteristics of the pain is the aggravation produced by physical movements of the thorax or twisting or turning the trunk, or associated with coughing, deep respirations, or sneezing. Frequently anxiety associated with the pain greatly adds to the confusion and has been known to result in a shocklike syndrome.[14]

Occasionally pleuropericarditis is an added source of pain. The pain usually lasts from one to three days. Many of the patients feel more comfortable in the erect posture.

The one most consistent physical finding which aids in the diagnosis is a pericardial friction rub. It may be heard within a few hours from the onset of chest pain, usually within the first day. A rub is heard in about 75 per cent of the cases. It is usually loudest in the third and fourth interspaces just to the left of the sternum. The presence of fever with the onset of the disease is the usual finding. Tachycardia is a common observation, usually with a pulse rate of 104 to 120 per minute.

Cardiac enlargement is one of the important and fairly consistent findings. There are divergent opinions as to the chief cause of the enlarged cardiac silhouette. Some think that the dilatation is due to a toxic myocarditis which results in a loss of tone and lengthening of the muscle fibers, but not to an extent to impair the functional capacity. Porter and others[14] believe that effusion explains it best since they doubt that the heart could be dilatated so much in the absence of gallop rhythm, heart failure, or serious arrhythmia and the patients consistently have complete recovery. Recent years have brought better roentgen-ray techniques to help settle this problem without a tap. The observations of the changes of cardiac configuration brought about by different positions of the patient can be used to differentiate between effusion and dilatation. In 1949 Williams and Steinberg[18] described a technique of angiocardiography whereby they were able to demonstrate a characteristic shadow due to pericardial fluid surrounding the opacified cardiac chambers following injection of dye. Pleural involvement is a frequent finding associated with pleural effusions.[3, 8, 13] In most instances there has been some moderate leukocytosis, and usually some degree of left shift. There is agreement that there is a consistently elevated sedimentation rate, usually elevated when the patient is first seen.

Electrocardiographic results are consistent and characteristic and aid in diagnosis. Vander Veer and Norris in 1937[17] correlated the electrocardiogram findings and the pathologic findings at autopsy in some cases of pericarditis which gave some of the earliest well established electrocardiographic patterns found in acute pericarditis. The characteristic pattern they found consisted of an elevation of the R-T segments in the three limb leads. It has been observed that the S-T segments are concave, in contrast to the convex or cove plane segments so often seen in myocardial infarction. A-V conduction times remain unchanged. Characteristic T wave changes occur[14] with lowering in amplitude, and there may be progression from an isoelectric level on to inversion. The T wave changes seem to appear later than S-T segment changes and return to normal more slowly. However, any and all of the changes may be transient and may not last more than a few days. In a few cases of acute benign pericarditis transitory arrhythmias have occurred.[8] These have been mostly frequent ventricular extrasystoles and supraventricular tachycardias. P-R interval prolongation has not been observed.

PERICARDITIS v. INFARCTION

Often in cases of benign pericarditis the clinical picture and pain description must be differentiated from many diseases involving the pleura, pericardium, and other thoracic structures, but the chief problem is deciding between it and acute myocardial infarction. As intimated in the name of the process, the outlook for these patients as compared to those with infarction is entirely different.[1] The diagnosis of acute myocardial infarction carries an immediate grave prognosis, a guarded ultimate prognosis, a prolonged convalescence, and a permanent modification of the patient's way of living. In contradistinction, a diagnosis of acute benign pericarditis intimates a good immediate as well as long-term prognosis, promises a period of convalescence usually much shorter, and allows the patient to return to his normal life after recovery from the acute process. The pain in pericarditis is seldom so agonizing as in infarction. The distinguishing feature is the accentuation of the pericarditis pain by bodily movements such as twisting or turning in bed, moving the trunk, coughing, or sneezing.[2, 9, 19] Often the patient with pericarditis is more comfortable in sitting, leaning forward, or erect posture and avoids the prone position. The opposite is true with infarction. Patients with pericarditis often have had antecedent respiratory infections and complain of general malaise and a "la grippe" feeling more than in cases of infarction. The pericardial friction rub in pericarditis is heard at or within a few hours of onset of pain, is heard over a wider area, is louder, and usually lasts longer than that associated with myocardial infarction. Cardiac silhouette enlargement can be a differentiating point, especially since it is unusual in acute myocardial infarction. Characteristic electrocardiographic patterns occur in both diseases and may be readily differentiated, especially with serial tracings. In general, the average age of patients with benign pericarditis is

lower than that of patients with myocardial infarction.[3, 8, 15]

Some clinicians feel that an entirely optimistic prognosis for these patients with benign pericarditis must be somewhat guarded,[6] but no instance of constrictive complications has been reported despite the fact that some patients have been followed for several years, some for more than sixteen years.

The 12 cases of benign pericarditis diagnosed at Dallas Veterans Administration Hospital have occurred in the last three years. It may be observed from table 1 that cases have a fairly wide range as to age and physical and laboratory findings. Present in these cases are symptoms and signs similar to the various ones described in the literature. Antecedent respiratory infections and pericardial friction rubs were found in about the usual ratio of cases (about two-thirds). Temperature elevations were about as others have observed. White blood cell counts varied a great deal. In 8 cases the white cell counts were elevated and 2 cases manifested an eosinophilia. Cardiac silhouette was found enlarged in 5 cases. As in most series, practically all cases manifested abnormal electrocardiographic tracings which returned to normal with recovery. Sedimentation rate was uniformly elevated in all. Pain description, radiation, and aggravating causes were generally the same as those characteristically described. Patient 4 had a partial pneumothorax for tuberculosis which had been inactive for eighteen months and no more air was given after the pericarditis illness. A two year follow-up revealed that the patient remained asymptomatic and worked regularly. A Negro patient (case 6) who was admitted in diabetic acidosis developed pericarditis, and friction rub persisted sixteen days, longer than in any other case. This patient and patient 11 early had a supraventricular tachycardia which disappeared in about two days. Pericardial taps were done in 2 cases but no effusion fluid was obtained. Aureomycin was given without obvious effect in 3 cases. Two cases had had, by history, similar episodes previously. The average age in the group reported here was essentially the same as in the total series of 197 cases.

SUMMARY

Benign pericarditis has been recognized for almost 100 years and is not a rare disease, but it often requires astute differentiation from more common diseases by careful history and observation.

Present concepts of etiology are discussed and although the cause is not definitely known, I favor a hypersensitivity reaction to previous infection, such as in rheumatic fever and glomerulonephritis.

Symptoms and clinical and laboratory findings are reviewed in the light of cases reported in the literature and in 12 additional cases which present a fairly characteristic set of manifestations.

The importance of distinguishing benign pericarditis and acute myocardial infarction is mentioned, and the chief differential points are given.

There has been no known specific therapy which consistently has shortened the course of the disease. Treatment, therefore, is symptomatic and supportive.

Travis Clinic.

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ABSTRACT OF DISCUSSION

DR. RICHARD DATHE, Dallas: Dr. Verheyden has obviously done a great deal of work in completely scanning the literature, in studying reported cases of benign pericarditis as well as his own cases, and in tabulating the characteristics of this disease.

Benign pericarditis must be considered in the differential diagnosis of any patient with substernal or precordial chest pain. Although this type of pericarditis is considered benign, and apparently is so since no deaths have been reported, the fact remains that pericardial effusion frequently develops and

BENIGN PERICARDITIS — Verheyden — *continued*

can accumulate with sufficient rapidity to cause serious cardiac tamponade. I know of 1 case in which pericardiocentesis was, I believe, life-saving. These cases should be carefully observed for this possibility.

The differentiation of benign pericarditis from acute myocardial infarction is extremely important and can be made as Dr. Verheyden has indicated. However, in the absence of pericardial friction rub or characteristic electrocardiographic changes, the diagnosis of benign pericarditis may be impossible in the first few days. In this event serial daily fluoroscopic or roentgenographic examination is unequalled in showing the progress of the disease, especially when pericardial fluid develops, producing rapid changes in the cardiac silhouette.

THROMBOANGIITIS OBLITERANS (Buerger's Disease) Role of Sympathectomy in Treatment

LeROY J. KLEINSASSER, M.D., Dallas, Texas

THE diagnosis of thromboangiitis obliterans presents a real problem even after the tissues are examined microscopically. Basically, the diagnostic criteria I employ are those set down by Buerger,[2] and others. Buerger believed that "for the clinical diagnosis of thromboangiitis we must depend upon: (1) the racial (Hebrew) and sex (male) predilection; (2) the early involvement of the lower extremities; (3) the early symptoms of pain or intermittent claudication; (4) the presence of migrating phlebitis; (5) the evidence of pulseless vessels; (6) the presence of blanching of the extremity in the elevated position; (7) the existence of rubor in the dependent position; (8) the relation of the hyperemic phenomena to posture; (9) the absence of simultaneous, symmetrical involvement; and (10) the slow, progressive chronic course terminating in gangrene." In addition, the patient must have the onset of symptoms before the age of 40 years and there should be no roentgenographic evidence of arterial calcification and no hypertension.

Using these criteria, I am reporting on 18 patients, all of whom had the onset of their vascular problems before the age of 40. The duration of the manifestations varied from three weeks to twenty years (table 1).

The principal symptoms (table 2) in the order of their occurrence initially were claudication in 6 patients, foot pain in 3, migratory phlebitis in 2, and cold sensitivity in 2. Although intermittent claudication occurred initially in 6 patients, ultimately it was reported by 15 patients. This severely restricted these patients' ability to walk. Intermittent claudication may be helped early by advising the patient to walk more slowly, but frequently the situation becomes severe. No patient in this group was able to walk more than four or five blocks without claudication.

Read before the Section on Surgery, Texas Medical Association, Annual Session, Dallas, May 7, 1952.

INDICATIONS FOR SYMPATHECTOMY

Claudication presents one of the major indications for sympathectomy in occlusive arterial disease. The sensation of coldness or the objective evidence of coldness of the involved extremity was common in this group and occurred in 10 patients. This sensation is partially related to the obviously diminished blood supply, but its subjective manifestations are difficult to explain. It is well known that patients who demonstrate or report a history of migrating superficial thrombophlebitis must be carefully observed for the development of Buerger's disease. Initially, this manifestation occurred in 2 patients, but 7 developed such changes during the course of their disease. The migratory superficial vein involvement most frequently appears to involve the branches of the internal (long) saphenous vein but may involve any superficial vein, including those of the upper extremity. Any patient

TABLE 1.—*Initial Symptom of Thromboangiitis Obliterans in 18 Patients and Duration of the Manifestations.*

Symptom	Cases
Claudication	6
Foot pain	3
Migratory phlebitis	2
Cold sensitivity	2
Legs go to sleep	1
Failure of ingrown toenail to heal after excision	1
Gangrene of finger	1
Gangrene after injury to knee	1
G. S. W. leg	1
Duration of symptoms	
4 years or less	11
7 to 9 years	4
10 years or more	2
(range: 3 weeks to 20 years)	

TABLE 2.—*Principal Symptoms in Order of Occurrence in 18 Cases of Thromboangiitis Obliterans.*

Symptoms and Signs	No. Cases
Intermittent claudication	15
Coldness	10
Ulceration	9
Migratory phlebitis	7
Edema	6
Raynaud's phenomenon	4
Pain in foot on activity	3

with such a history should be examined for arterial involvement.

An interesting finding is the history of Raynaud's phenomenon, usually in one extremity in contrast to the classical vasospastic form of the disease, which is symmetrical in occurrence and without evidence of occlusive involvement of arteries. This is the primary differential diagnostic point. It is well known that a considerable number of vascular disorders present the vasospastic phenomenon of Raynaud's disease in the form of color change in some stage of their development, and primary causes of such phenomenon must be sought. Edema is not a prominent manifestation but frequently may occur locally in an area of acute involvement. Rarely is it a significant finding unless it is pronounced with evidence of inflammation. The pain in the foot is characteristically described as stinging, burning, or as the sensation of walking on gravel. It must be cautioned that rarely are arch supports in shoes indicated under such circumstances, and they may even be harmful.

Smoking presents a real problem in patients with thromboangiitis obliterans and 11 admitted smoking from 1 to 3 packages of cigarettes per day. In a review of 1,400 cases Silbert[5] stated that without exception all patients with thromboangiitis obliterans had been tobacco smokers. He even doubted the diagnosis of Buerger's disease if the patient gave no history of smoking. He stated that the disease is uniformly progressive in persons who continue smoking and immediately and completely arrested in those who permanently discontinue smoking. Based on this information and upon personal observation, I have attempted to persuade patients to stop smoking. Anyone who has smoked knows that the habit is difficult to overcome. This is especially true in the patient with thromboangiitis obliterans, who probably also has some visceral and cerebral vascular involvement. The patient may even hide the cigarettes in the bedside table and state that he does not smoke. The extreme example of this is a patient I once saw who lost all his extremities with this disease while acknowledging that the discontinuance of smoking was associated with a remission of his disease. The last time I saw him he had a table holder for a cigarette with a connecting tube to his mouth so that someone could place the cigarette into the holder and light it for him. This merely illustrates the seriousness of the problem. I believe there is no question that patients with thromboangiitis obliterans should be advised against smoking, yet there seems to be no real unanimity of opinion on this matter among physicians.

In establishing the diagnosis of thromboangiitis obliterans, the presence or absence of arterial pulsations is significant. It is imperative that the following pulses be felt carefully: the dorsalis pedis, posterior tibial, popliteal and femoral in the lower extremities, and radial and ulnar in the upper. The ulnar pulse is determined by inference as described by Allen.[1] Since thromboangiitis obliterans is an arterial occlusive disease, there should be objective evidence of its occurrence. There is also the associated evidence of arterial insufficiency, as manifested by pallor on elevation of the extremities, rubor on dependency, and frequently a delayed venous filling time. Although most of these patients were studied plethysmographically (digital) and thermometrically, these determinations are not absolutely essential for this purpose, as palpation and examination will evaluate properly the evidences of impoverished blood supply.

In an attempt to evaluate the degree of vasospasm and possible response to sympathectomy, regional sympathetic blocks[4] with procaine were done preoperatively on all patients. This determination is not entirely essential since there may be little change in the skin temperature. However, the procedure can be used prognostically in that if there is much rise in skin temperature, there is indication of vasospasm, and sympathectomy would be encouraged in an attempt to (1) alleviate claudication, (2) heal ulceration or minor cutaneous gangrene, or (3) overcome the effects of sudden thrombotic occlusion. A lack of rise in skin temperature with sympathetic regional block with procaine is not a contraindication to sympathectomy, whereas a fall in skin temperature appears to be a definite contraindication. Based on thermometric, plethysmographic, objective, and symptomatic response, good effects from the regional blocks were observed in 14 patients. None demonstrated a fall in temperature; 3 noted relief of pain; and 4 experienced increased ability to walk.

In general, these patients had been treated with vasodilators, whiskey, sympathetic blocks, Buerger's exercises, and numerous other means of therapy. However, they were considerably disabled and sympathectomy was done as soon as possible. I believe that conservative measures should be tried when possible, but one should not persist in their use when indications are present for more aggressive therapy of sympathectomy or amputation. It is my belief that sympathectomy does not alter the course of the disease and should be performed only when indicated by the following conditions:

1. Thrombotic occlusion where no gangrene is present.
2. Intermittent claudication.
3. Minor ulcerations and limited areas of cutaneous gangrene.

I recognize that some physicians recommend bilateral sympathectomy when the diagnosis is estab-

lished and the indications for sympathectomy are present in either extremity. Since I believe that sympathectomy does not affect the course of the disease, which consists of either gradual arterial occlusion or, more frequently, episodes of thrombotic occlusion which may be disastrous, I have limited the sympathectomy to the extremity symptomatically involved. The one danger inherent in this plan is the patient's failure to return if the other extremity becomes symptomatic.

RESPONSE TO SYMPATHECTOMY

In this group of 18 patients, of whom 7 had bilateral lower extremity involvement and one quadrilateral involvement (table 3), 19 lumbar and 2 preganglionic dorsal sympathectomies[6] were done based on the foregoing criteria and indications. Fourteen patients had good end results from their sympathectomy in the preservation of the extremity and relief of symptoms. Twelve patients experienced significant relief of intermittent claudication (table 4). Sometimes this response to sympathectomy is delayed for several months, but ultimately there appears to be excellent improvement. Follow-up studies from one to five years seem to indicate that, once the vascular situation becomes stabilized after sympathectomy, there is a prolonged and slow period of improvement. One patient examined recently who had been operated upon one and a half years ago is typical in this regard. He had the onset of his symptoms six years before, and upon recent examination had pallor on elevation and rubor on dependency of the asymptomatic foot, but good color on elevation of the denervated extremity. Of 9 patients with varying degrees of cutaneous ulceration, 6 had satisfactory healing of the lesions. Three patients required minor amputations of the toes and 1 required amputation for gangrene of a finger. In no instance was sympathectomy effective in altering the course of the disease where there was a significant amount of gangrene present in the foot.

TABLE 3.—*Extremities Involved in 18 Patients with Thromboangiitis Obliterans.*

Extremities Involved	No.
Upper	4
Lower	23
Bilateral	7
Quadrilateral	1

TABLE 4.—*Results of Sympathectomy in 18 Cases of Thromboangiitis Obliterans.*

		No. Cases
Relief of claudication		12
Healing of ulceration (cutaneous)		6
Extension of manifestation to other extremities		4
Amputations		8
Minor	4	
Major	4	

Ultimately, 4 amputations below the knee were required and in 1 of these patients it was necessary to do a secondary amputation above the knee. All amputations were closed and it is possible that a guillotine type of amputation would have been desirable in the one failure of a below-the-knee amputation. In addition, 1 patient has returned with complete relief of symptoms following sympathectomy for pain in the left lower extremity. However, color changes were apparent in his right foot for the preceding eight months, and despite instructions to return at once for sympathectomy upon the extremity, he failed to return until there was considerable gangrene of his toes. Sympathectomy was done at once but was ineffective and an amputation below the knee was required. These are considered failures of sympathectomy, and one represented acute progressive arterial involvement with gangrene of the foot despite sympathectomy.

CASE REPORTS

CASE 1.—W. L. B., a white man, aged 43, nine years ago had the onset of muscular soreness in the calf of the right leg which became worse after exertion or walking far and required rest for relief (intermittent claudication). Eight years ago varicosities in the right leg were injected. Six years ago the left lower extremity became similarly involved and the patient developed numbness related to the intermittent claudication, which became progressively worse. During this period he smoked one package of cigarettes a day. Two or three weeks before admission of the patient to the hospital on July 22, 1950, the condition became severe in his left leg with much pain requiring morphine for relief. His blood pressure was 140/80. Femoral artery pulsations were evident. The left foot was cooler, blanched more on elevation, and demonstrated some cyanosis in the nail beds.

Lumbar sympathetic block with procaine was done with slight subjective improvement and increased walking distance. A left lumbar sympathectomy was performed under spinal anesthesia on July 31, removing the first, second, and third lumbar ganglia. Plethysmographic (digital) and thermometric studies during operation demonstrated no improvement. The patient was given some vasopressor substance to control his hypotension at the end of operation. Upon his return to the ward, the extremity was noted to be cold, cadaveric, and cyanotic. Priscoline given intravenously gave no aid. The condition of the extremity gradually improved so that by August 1 there was a slight elevation of temperature on the left side. The following day he developed tenderness in the left calf. The cyanosis continued, but the pain disappeared. On August 15 the patient was sent home, but he soon returned with a shallow ulcer over the left hallux with mild pain. The situation rapidly progressed with severe pain, and on September 28 an amputation below the knee was done with spinal anesthesia. Postoperatively he became disoriented and the wound failed to heal. An above-knee amputation was done November 2 and the skin left open but the fascia closed. By December 14 the wound was completely healed. Three months later the stump was in good condition, a satisfactory prosthesis was applied, and the patient became ambulatory.

A progressive lesion as represented in this patient calls for amputation without preliminary sympathectomy. I now

follow this procedure, which accounts for the low number of amputations following sympathectomy. Actually I believe the disease was aggravated by the sympathectomy, indicating that we did not properly evaluate the situation.

CASE 2.—M. J. B., a white man, aged 33, stated that his difficulty began ten years previously following a slight injury while in military service. He would experience pain in his knee on walking four blocks. Seven years ago his left ankle began to swell and became red and tender for several weeks. He smoked one package of cigarettes a day. Three to four months before admission to the hospital he developed an intermittent cramping in the calf of the leg brought on by exercise and relieved by rest. His blood pressure was 120/80. There was coldness of the left foot with slight pallor, and no peripheral pulses were noted. There was pallor on elevation of the left foot as well as tenderness in the calf. The venous filling was delayed, and on dependency the pallor persisted. There was some slight involvement on the right. A femoral arteriogram,[8] visualized no vessel below the knee. On November 8, 1950, a left lumbar sympathectomy was done removing the first, second, and third lumbar ganglia. There was a slight rise in skin temperature postoperatively, but this cooled, and there was a definite demarcation of warmth and coolness of the foot. The pain became worse. Amputation was advised November 19 because of severe pain, and the operation took place three days later. By January 9, 1951, his rehabilitation was complete with shrinking of the stump followed by active use of a prosthesis.

CASE 3.—W. C. A., a 29 year old white man, was a patient who required subsequent amputation. He had the onset of claudication of the right leg muscles and ulceration of the right hallux. A lumbar sympathectomy was done August 4, 1949, with healing. The man was warned to stop smoking but he persisted. By April 26, 1951, his symptoms in the left lower extremity became prominent, and a left lumbar sympathectomy was done. He was seen again September 26 with severe pain in his left foot requiring much morphine and being only partially relieved. He developed cyanosis in the foot and amputation below the knee was done. He was still smoking one package of cigarettes a day immediately postoperative.

TECHNIQUE

The technique of sympathectomy in the upper extremity is the preganglionic dorsal procedure as described by Smithwick.[6] In the lower extremity I employ a transverse incision from the tip of the eleventh rib to the middle of the rectus sheath, cut the anterior rectus sheath and aponeurosis of the external oblique muscle as well as the muscle laterally. The lateral edge of the posterior rectus sheath is also divided. Laterally the internal oblique and transverse muscles are split after incising the overlying fascia. An extraperitoneal exposure of the sympathetic ganglia then is made and usually the second and third lumbar ganglia are removed with the intervening chain. If the vascular involvement extends to the femoral arteries, the first lumbar ganglion is also removed.

CONCLUSIONS

It must be emphasized that the sympathectomy does not alter the course of the disease but does help to control its effects. If the arterial occlusion is gradual, there is a tremendous spontaneous growth of collaterals. Claudication may result requiring sympathectomy to stimulate further the growth of collaterals.

Should there be a sudden episode of thrombotic occlusion, the situation is much more serious and the problem is not only one of collaterals but a widespread vasospasm. If more conservative measures of vasolidatation by regional sympathetic block are not immediately effective, sympathectomy may become an urgent necessity.

I wish to caution that sympathectomy should not be done needlessly in the faint hope of aiding an irretrievable situation when actually amputation is required. The contraindications to sympathectomy are:

1. Acute gangrene (especially if progressive).
2. Severe pain unrelieved by conservative measures and requiring narcotics for relief.
3. Fall in skin temperature after administration of regional sympathetic block.

SUMMARY

Eighteen patients on whom 2 cervicodorsal sympathectomies and 19 lumbar sympathectomies were done for thromboangiitis obliterans are reported.

Four required subsequent major amputations and 4 required minor amputations.

The procedure is effective for claudication, widespread vasospasm of the extremity, and minor ulcerations and areas of cutaneous gangrene.

It is believed that sympathectomy should be limited strictly to the proper indications in the extremity that is symptomatically involved.

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ABSTRACT OF DISCUSSION

DR. MICHAEL E. DE BAKEY, Houston: This presentation by Dr. Kleinsasser, based upon his experience with 18 cases of thromboangiitis obliterans, constitutes a thoughtful and critical consideration of the problem. He has placed proper emphasis upon certain important features of the problem, particularly as they concern therapy. For the most part my experience is in accord with his observations.

The importance of tobacco smoking as an aggravating

THROMBOANGIITIS OBLITERANS — Kleinsasser — *continued*

factor in this disease has been well established. Like others I have frequently observed almost complete regression of symptoms following abstinence of tobacco smoking and recurrence with resumption of smoking. I am firmly convinced that no form of therapy is effective unless there is complete and permanent abstinence of tobacco smoking.

Most observers with extensive experience with this disease are now agreed that sympathectomy is the treatment of choice and offers the best means of improving the circulation. In my experience the best results are obtained in the slowly progressive type with moderate degrees of involvement. Much less satisfactory results have been observed in the acute fulminating and rapidly progressing type of the disease. In fact this severe form of the disease, which fortunately occurs less frequently than the other type, has been found resistant to all forms of therapy including ACTH and cortisone. Contrary to Dr. Kleinsasser's observation, however, I have not encountered instances of aggravation of the disease by sympathectomy.

There is still some controversy concerning the best technique of sympathectomy or what constitutes complete denervation, particularly for the upper extremity. Dr. Kleinsasser has indicated his preference for the preganglionic dorsal procedure. This method was originally proposed by Freeman, Smithwick, White, and Ascroft*,†,‡ on the basis that it would prevent degeneration of the postganglionic neurons and the resultant hypersensitization of the denervated vessels to circulating epinephrine. This was believed to be an important factor in limiting the enduring effectiveness of sympathectomy particularly in Raynaud's disease. Considerable experience has since accumulated, however, to show that the results of preganglionic sympathectomy in these cases were no better than those of postganglionic sympathectomy and that return of sympathetic activity occurred with equal, if not greater, frequency after the preganglionic operation. For this reason I have abandoned the preganglionic type of operation and now employ ganglionic sympathectomy which I believe provides a more complete denervation of the part. I also prefer the anterior approach for the upper extremity because it may be performed with less extensive surgical trauma than the dorsal approach.

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‡*Smithwick, R. H.; Freeman, N. E.; and White, J. C.: Effect of Epinephrine on Sympathectomized Human Extremity; Additional Cause of Failure of Operations for Raynaud's Disease, Arch. Surg.* 29:759-767 *(Nov.) 1934.*

INDICATIONS FOR SPLENECTOMY

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THERE are, at times, certain definite indications for splenectomy, such as when the spleen has ruptured and there is bleeding into the abdominal cavity. However, the indications are not always so definite and the physician is uncertain as to what results to expect from removal of the spleen. The intent of this review is to outline the indications and contraindications for splenectomy and to clarify the predictions as to results.

The functions of the spleen are but incompletely understood. It is intimately concerned with blood destruction and potentially with blood formation. In fetal life the spleen is active in the formation of all types of cells, and it can revert to this function upon demand at any period of life. In the adult, this activity is limited to the production of lymphocytes and monocytes. The spleen also actively participates in the elimination of erythrocytes. This normal process of red blood cell destruction is greatly increased in certain pathologic conditions. In addition, the spleen may acquire an abnormal phagocytic ability in respect to white blood cells and platelets.

This abnormal splenic phagocytosis is the concept of hypersplenism espoused by Doan and his co-workers.[6] However, Dameshek and others[5] maintain that the spleen normally exerts an inhibitory effect on the bone marrow and that this function may be exaggerated when splenomegaly occurs, resulting in the syndrome of hypersplenism. Evans and others[8] believe that regardless of whether decreased formation or increased destruction best explains the depression of the cellular elements of the blood, the spleen alone is not the cause of this reduction. With particular reference to thrombocytopenia and acquired hemolytic anemia, they postulate the presence of an auto-antibody for red blood cells and thrombocytes. These writers believe that the principal role of the spleen lies in its function as an antibody-producing tissue rather than through direct destruction.

Regardless of the underlying mechanism involved, from the standpoint of surgical intervention and removal of the spleen, there are rather standard classifications according to the disease syndromes.[1,2] In the first group, splenectomy is usually alleviative or curative (table 1). In the second group, splenectomy is indicated only in selected cases (table 2). In the third group, splenectomy is contraindicated either for the reason that it is not beneficial or that it may produce harmful effects (table 3).

Six of these disease syndromes with exemplary cases are presented.

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CONGENITAL HEMOLYTIC JAUNDICE

Congenital hemolytic jaundice occurs in any race, in either sex, and at any age. The disease is not only congenital but is familial as well.

The diagnostic triad is anemia, jaundice, and splenomegaly. However, any or all of these may be absent, which indicates the extreme variability of the disease and the fact that the spherocytosis and increased fragility of the red blood cells are the basic diagnostic findings in all cases.

TABLE 1.—*Conditions in Which Splenectomy Definitely Is Indicated.*

Traumatic conditions of the spleen, rupture, hemorrhage.
Ptosis of the spleen with torsion of its pedicle.
Primary splenic tumors—giant follicular lymphoblastoma, lymphosarcoma, endothelioma, and reticulum cell sarcoma.
To aid in exposure in radical operations in the left upper quadrant of the abdomen.
Congenital hemolytic anemia.
Acquired hemolytic anemia (primary).
Idiopathic cytopenic purpura.
Primary splenic neutropenia or panhematopenia.
Banti's syndrome, congestive splenomegaly.
Thrombosis of the splenic vein.
Massive splenomegaly producing pressure symptoms.
Splenomegaly of unknown origin.

Modified from Limarzi, L. R.; Sloan, L. H.; Puestow, C. B.; and Cole, W. H.: Indications for Splenectomy, Am. Pract. *4*:25-33 (Sept.) 1949.

The diagnosis is established in the presence of many microcytes in the blood smear with a normal mean corpuscular volume of 80 to 90 cubic microns. The blood hemolysis begins usually at .48 and is complete at .42 per cent salt solution, whereas normally hemolysis begins at .42 and is complete at .34 per cent. The jaundice can readily be differentiated from obstructive jaundice by the presence of normal or increased bile pigments in the stools and its absence in the urine (acholuric jaundice). Examination of the stools often will reveal excess amounts of urobilinogen.

TABLE 2.—*Splenectomy Indicated Only in Selected Cases.*

Secondary panhematopenia.
Felty's syndrome.
Tuberculosis of the spleen.
Boeck's sarcoid.
Syphilitic splenomegaly.
Hodgkin's disease.
Cirrhosis of the liver.
Leukemia.
Gaucher's disease.
Aplastic anemia.
Benign cysts and tumors.
Abscess of spleen.

Modified from Limarzi, L. R.; Sloan, L. H.; Puestow, C. B.; and Cole, W. H.: Indications for Splenectomy, Am. Pract. *4*:25-33 (Sept.) 1949.

From the standpoint of differential diagnosis, congenital hemolytic anemias must be separated from a large group of acquired hemolytic anemias either secondary or primary.

Secondary hemolytic anemias may result from circulatory hemolysins present following repeated transfusions of an Rh negative patient with Rh positive blood, after activation of hemolysis in syphilitic persons by exposure to cold, or as a result of burns, some infections, or snake venom. Malaria, heavy metal poisoning, and sensitivity to drugs, including Pyribenzamine and Benadryl,[4] are other etiologic factors to be considered. Hemolytic anemias may be a complicating feature of many diseases, such as leukemia, pernicious anemia, and severe liver damage. However, the underlying disease is usually predominant and fairly readily recognized. In these cases, splenectomy would not be expected to improve the basic underlying condition.

TABLE 3.—*Conditions in Which Splenectomy Is Contraindicated.*

Polycythemia vera, Vaquez-Osler disease.
Agnogenic myeloid metaplasia of the spleen.
Cooley's anemia.
Sickle cell anemia.
Metastatic malignant tumors.
Acute splenic tumors; infection.

Modified from Limarzi, L. R.; Sloan, L. H.; Puestow, C. B.; and Cole, W. H.: Indications for Splenectomy, Am. Pract. *4*:25-33 (Sept.) 1949.

The criteria of diagnosis for primary acquired hemolytic anemia include the following:

1. The presence of anemia with reticulocytosis, increased bilirubin, and increased fecal urobilinogen.
2. Absence of family history of hemolytic disease.
3. Immunologic evidence of sensitization of red blood cells by an antibody active at 37 C. (Coombs' test).
4. Rapid destruction of transfused cells.

When the diagnosis of either congenital hemolytic icterus or primary acquired hemolytic anemia is established, splenectomy is advisable. Blood transfusions in such patients often are followed by a severe reaction, even when blood matching has shown no incompatibility. Accordingly, it is generally agreed that blood transfusions should be started, if possible, only at the end of the operation after the splenic pedicle is ligated.

CASE 1.—A 22 year old Latin-American housewife, E. Z., was first seen July 8, 1949, with a chief presenting complaint of gastrointestinal disturbances for two weeks following a trip to Mexico. Past history revealed that she had had a constant, though varying degree of jaundice since childhood. She was studied at the Robert B. Green Hospital eighteen years before, at which time she was told that she had liver disease. The records were not available.

Examination revealed an active, alert girl who appeared no sicker than anyone else with a mild gastroenteritis. The skin and mucous membranes were pale, waxy, and definitely icteric. The gastrointestinal symptoms cleared promptly under the usual therapy.

Laboratory studies revealed a red blood cell count of 3,180,000, hemoglobin of 64 per cent, and a white cell count of 6,500 with a normal differential. The blood smear revealed many microcytes. The hematocrit level was 29 cc.

per 100 cc. of blood; the mean corpuscular volume 91 cubic microns. The serum bilirubin, direct was 1.55 mg. per 100 cc. and indirect was 8.3 mg. The urine was negative for bile, with a urobilinogen positive up to a 1:10 dilution. The stool was bile positive, with a fecal urobilinogen of 96 mg. The fragility test revealed a beginning hemolysis at .50 per cent salt solution with complete hemolysis at .44 per cent. The bromsulphalein test revealed a 3 per cent retention of dye in thirty minutes. The cephalin flocculation was 2 plus.

This patient has had an apparently normal life and splenectomy, which was recommended but not urged, was refused.

IDIOPATHIC THROMBOCYTOPENIC PURPURA

Idiopathic thrombocytopenic purpura is a disease that may be acute or chronic and is characterized by a marked and unaccountable diminution in the number of platelets and spontaneous hemorrhages from the mucous membranes and skin and in some cases the bowel, kidneys, vagina, and brain.

Young persons are more often affected than old ones; females are more often affected than males.

Examination, in addition to the hemorrhagic signs, usually reveals a slight enlargement of the spleen. Marked enlargement of the spleen is not usually found in thrombocytopenic purpura and suggests some other diagnosis.

The pathogenesis of primary thrombocytopenic purpura has not been satisfactorily explained. Humble[10] noted increased permeability of the arteriolar end of the capillary loop in 5 cases of this disease and postulated a selective poisoning at this junction. Allen and Jacobson[1] demonstrated the presence of increased heparin-like activities in cases with purpura and reported the use of toluidine blue and protamine in neutralizing this effect.

Fundamentally, however, the disease is due to a reduction of platelets. This reduction, according to Dameshek and others,[20] is the result of an exaggerated function of the spleen or hypersplenism. Accordingly, there is a block in the delivery of the marrow cellular elements (in this case the thrombocytes) to the peripheral blood, and perhaps in addition, there is increased phagocytosis of these cells by the enlarged spleen.

Evans[8] demonstrated an antithrombocyte antibody in the serum of patients with thrombocytopenic purpura. He expressed the belief that acquired hemolytic anemia and thrombocytopenic purpura are comparable in the underlying pathogenesis and that the primary role of the spleen in this condition lies in its function as an antibody-producing tissue. When splenectomy is successful, it appears that antibody production has been reduced below a critical level. Such a concept would help to explain a patient with idiopathic thrombocytopenic purpura reported by Yates[23] who did not respond to splenectomy, who did develop a lymphatic hyperplasia postoperatively, and who did not have accessory spleens at autopsy.

The course of thrombocytopenic purpura is one of spontaneous remissions. The course may be extremely mild, particularly in infants and persons over 40 years old, when splenectomy is rarely indicated. In the acute bleeding phases, operation carries a serious risk. At such times, the patient should be supported with transfusions until a remission occurs, when splenectomy may be performed much more safely. Occasionally, it will appear obvious that medical management is ineffective and an emergency operation is indicated.

The results of splenectomy in this condition are 80 per cent favorable. In some cases, there is a return of the platelet count to the preoperative levels, yet the tendency to bleed is much less. In a certain number of patients, permanent improvement does not follow removal of the spleen. These failures following splenectomy have been attributed to the presence of an accessory spleen or a permanent failure of the platelet-forming mechanism for some unknown cause. Removal of the accessory spleen in the first instance will result in a permanent cure. In the second instance, repeated blood transfusions may carry the patient along until the failure of the platelet-forming mechanism has been relieved.

CASE 2.—A 22 year old white housewife had noted easy bruising at the age of 6 years. Menarche, with profuse and persistent menorrhagia, began when she was 14 years of age. Two months thereafter, examinations revealed numerous petechial hemorrhages over the legs, abdomen, chest, and mucous membranes of the throat and mouth. The spleen was barely palpable.

The red blood cell count was 4,520,000; hemoglobin, 12.7 Gm.; white blood cell count, 5,900, with 68 per cent neutrophils; platelets, 19,000 to 27,000; and bleeding time, twenty-six minutes.

A spleen and two accessory spleens were removed. Subsequently, the thrombocyte count has ranged from 50,000 to 60,000; red blood cell count about 3,500,000; and hemoglobin 50 to 60 per cent. The white blood cell count has remained within normal range. The bleeding time has been only slightly prolonged at four to five minutes with complete clot retraction occurring in eighteen hours. A bone marrow study made in 1948 was normal.

The patient continued to have petechiae and varying degrees of menorrhagia until onset of pregnancy in February, 1951. She tolerated pregnancy well but required transfusions every one to two weeks to maintain a red blood cell count averaging 4,000,000 and hemoglobin of 78 per cent, with a platelet count of from 56,000 to 92,000. Serum bilirubin in the last trimester measured direct, 1.5 mg.; indirect, 3.8 mg. A normal full term delivery was accomplished with supportive fresh blood transfusions before and after delivery.

Comment: This patient satisfies the criteria for diagnosis of idiopathic thrombocytopenic purpura, namely:

1. The presence of purpura, prolonged bleeding

time, and diminished clot retraction in patients with low thrombocyte count.

2. The observation of normal or increased megakaryocytes in the bone marrow.

3. Lack of evidence that thrombocytopenia is due to exogenous toxic agents or secondary to disease known to be associated with thrombocytopenia. Two drugs recently implicated in the production of thrombocytopenic purpura are prophylthiouracil[9] and digitoxin.[2]

PRIMARY SPLENIC HEMATOPENIA

Primary splenic hematopenia is characterized by fever, pain over the splenic region, and splenic enlargement. The course of the disease may be acute, subacute, or chronic in different cases. The blood picture will show a reduction of the white blood cells, red blood cells, or platelets, either singly or in any combination.

The bone marrow is normal or even hyperplastic. If bone marrow studies reveal a depression of the cellular elements, beneficial effect of splenectomy is doubtful and a search should be made for toxic or chemical source of the depression. Newer drugs that have been implicated in bone marrow depression are Mesantoin,[21] propylthiouracil,[9] Tapazole,[3] Pyribenzamine,[14] Chloromycetin,[19] streptomycin,[16] and mercurial diuretics.[18]

CASE 3.—A 26 year old white housewife presented a history of acute onset of nausea and vomiting of nonbloody material seven days prior to admission to the hospital. Following this episode she felt extremely weak and unable to get out of bed. One month previously she had been hospitalized for observation for an acute pain in the right lower quadrant of the abdomen. The symptoms subsided rapidly. At that time the red blood cell count was 4,190,000; hemoglobin, 11 Gm. or 74 per cent; the white blood cell count, 5,500 with 55 per cent neutrophils.

Physical examination revealed a well developed but pale and slightly icteric white woman who appeared acutely ill. The temperature was 102 F.; pulse rate, 120 per minute; respiration, 32 per minute; and the blood pressure, 160/56 mm. of mercury. A grade 2 systolic murmur was heard over the cardiac apex. The spleen was firm, smooth, nontender, and enlarged four fingers below the left costal margin and extending across the abdomen to the midportion of the right upper quadrant.

The red blood cell count was 1,350,000; hemoglobin, 26 per cent; white blood cell count, 1,750 with 51 per cent neutrophils; and platelets, 64,000. The icteric index was 31; the urine bile negative; and stools, bile positive with 480 mg. of fecal urobilinogen per 100 Gm. of stool. The bone marrow was normal. Smears for malaria were negative. An Adrenalin test[22] produced the following effect in thirty minutes: Platelets rose from 64,000 to 172,000; red cells from 1,850,000 to 2,820,000, and white blood cells from 1,750 to 2,400.

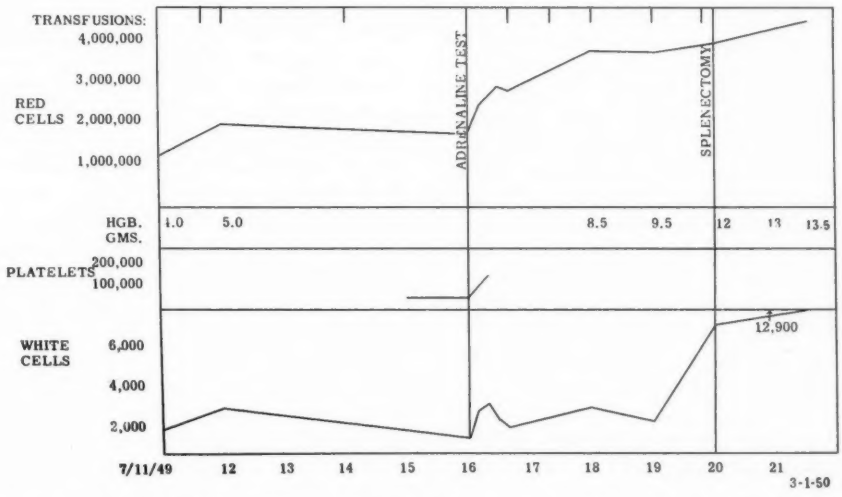

FIG. 1. The changing blood picture in a case of splenic panhematopenia (case 3) prior to and after splenectomy.

After four transfusions the patient's general condition improved, fever subsided, the red cell count was 3,870,000, and the white cell count 2,550 with 60 per cent neutrophils. The day following splenectomy the blood counts were 4,090,000 and 12,900 respectively. Eight months later, the patient developed hyperthyroidism. The red blood cell count at that time was 4,500,000 with 87 per cent hemoglobin, and the white cell count 14,000 with 40 per cent neutrophils. Thyroidectomy was accomplished uneventfully.

Figure 1 depicts graphically the changing blood picture in this case.

Comment: In addition to depression of all of the cellular elements in the peripheral blood, this patient also presents evidence of a hemolytic anemia as indicated by the presence of jaundice and an increased fecal urobilinogen.

SECONDARY SPLENIC HEMATOPENIA

Syndromes in which the etiology of the splenomegaly can be determined are referred to as secondary neutropenia or panhematopenia. Hypersplenism is a functional, not an anatomic diagnosis. Whenever the spleen becomes anatomically enlarged, whatever the cause for the enlargement, it may become physiologically hyperactive. In selected cases of secondary hypersplenism, removal of the spleen may be expected to result in a cure of the hematologic abnormality and the signs and symptoms attributable to the various types of cytopenia, even though the fundamental disease is not affected. Beneficial results of splenectomy have been described in Felty's syndrome, isolated Hodgkin's disease, Boeck's sarcoid, tuberculosis of the spleen, gummas of the spleen, Gaucher's disease, cirrhosis of the liver, and leukemia. Malignant nature of the underlying disease process does not necessarily contraindicate splenectomy. In a recent discussion, Sacks[15] described a patient with lymphosarcoma whose remaining life was made much more bearable by splenectomy. Several observers have recommended splenectomy in selected patients with leukemia on the basis of complicating hypersplenism.[11] This may be manifested by any of the four forms of hypersplenism. The hemolytic anemia is usually the most serious because it is the earliest.

The following laboratory findings were considered of value in indication for splenectomy in leukemia:[11]

1. A falling red blood cell count not reversible by transfusion except in excessive amounts.
2. Elevation of the reticulocyte count.
3. Lowering of the circulating leukocyte level (the granulocytic leukemia usually had a rise in the young forms).
4. Reduction of the platelets.
5. Hyperplasia of the bone marrow elements that were reduced in the peripheral blood, with the exception of lymphatic leukemias.

The following case is of splenomegaly of unknown origin which demonstrated the syndrome of secondary hypersplenism.

CASE 4.—Mrs. J. H. V., a 57 year old white woman, was first seen in September, 1950, with a history of an anemia not responding to administration of liver and iron for three months, accompanied by a weight loss of 15 pounds, and "hurting" in the left midabdomen for one week.

Physical examination revealed a moderately obese, not acutely ill-looking woman, with pallor of the skin and mucous membranes. Examination of the abdomen revealed a splenic mass involving the entire left upper half of the abdomen.

Hematologic studies on September 13 revealed the following: red blood cell count, 3,440,000; hemoglobin, 64 per cent; white blood cell count, 3,950; hematocrit level, 32 per cent; segmenters, 53 per cent; stabs, 6 per cent; lymphocytes, 32 per cent; monocytes, 8 per cent; and platelet count, 149,000.

After careful observation and studies, including bone marrow examination, serum bilirubin tests, gastrointestinal roentgenograms, and liver function tests, all of which were normal, and in the presence of a persistent panhematopenia, splenectomy was done. Preoperatively, after several transfusions, the blood count was as follows: red cells, 3,830,000; hemoglobin, 87 per cent; white cells, 5,350; and platelets, 38,300. The pathologic diagnosis was giant follicular lymphoblastoma. Two months thereafter, the red blood cell count was 4,520,000; hemoglobin was 90 per cent; white blood cell count was 6,350; and platelets were 254,700.

Comment: This patient presents an indication for splenectomy on the basis of splenomegaly of unknown origin, primary splenic tumor, and a potentially systemic disease in which the presenting symptoms are those primarily the result of hypersplenism.

BANTI'S SYNDROME (OR SPLENIC ANEMIA)

Banti's disease as a clinical entity has always been open to question. The syndrome is characterized as a chronic disease of unknown origin, primarily in the spleen, and accompanied by splenomegaly, anemia, leukopenia, a tendency to gastric hemorrhage, and increased destruction of blood cells. This is followed in the later stages by cirrhosis of the liver, esophageal varices, ascites, and jaundice. According to one view, the process results from a variety of lesions which produce hypertension in the splenic vein, causing a congestive splenomegaly. This extrahepatic block (cavernomatous transformation or atresia of the portal veins) may be congenital or the result of thrombosis of the splenic vein or one of its tributaries, either spontaneous or due to trauma or infections. Intrahepatic block may be caused by portal cirrhosis or thrombosis of the hepatic veins. When the syndrome arrives at the point of cirrhosis of the liver, the question arises as to which came first. There is little clinical or hematologic distinction between patients with extrahepatic or intrahepatic block. However, a high retention on the bromsulphalein test, a reversal of the albumin-globulin ratio, and a positive cephalin flocculation reaction all point to a diagnosis of cirrhosis of the liver with intrahepatic block. If the tests are negative, it is assumed that the block is extrahepatic. Under these circumstances, particularly in a young person in whom Hodgkin's disease, Gaucher's disease, leukemia, malaria, or some other specific infection cannot be definitely diagnosed, it is probably fair enough to apply the label of Banti's disease. There is considerable difference of opinion as to the benefit of splenectomy, but some investigators think that if the spleen is removed early enough, the course of the disease will be retarded. Recently, shunting operations to prevent hemorrhage from esophageal varices, to

reduce portal hypertension, and to improve hepatic function have been successfully performed by several investigators. This procedure is most easily accomplished at the time of splenectomy, at which time the splenic veins can be preserved for anastomosis with the renal vein. Linton and others[13] found end to side splenorenal anastomosis with preservation of the kidney to be the most desirable.

CASE 5.—C. B., a 45 year old book dealer, a white man, was admitted to the hospital with a history of severe gross hematemesis, with no preceding gastrointestinal complaints except for jaundice at the age of 15 years. He did have a past history of recurrences of left sided hemiplegia in 1931, 1936, and 1939, presumptively on the basis of a congenital aneurysm.

Physical examination revealed a normally developed but pale, acutely ill looking middle aged white male. There was neurologic evidence of an old left sided hemiplegia. Otherwise, no abnormalities were detected.

On admission the red blood cell count was 2,990,000; hemoglobin was 61 per cent; platelets were 160,000; and white blood cell count was 14,900 with 85 per cent neutrophils. The bleeding, clotting, and prothrombin times were normal. The bromosulphalein test was normal; cephalin flocculation was 2 plus. Roentgen ray and esophagoscopy revealed esophageal varices.

After three weeks of observation, during which time bleeding persisted, a resection of the lower one-third of the esophagus and splenectomy were done. The patient had no recurrence of symptoms for approximately one year thereafter, at which time he died following the fourth cerebrovascular accident. Autopsy revealed a congenital anomaly of the cerebral vessels. Unfortunately, no description of the portal vessels was recorded.

Comment: There was little evidence of liver dysfunction in this case and it might have been ideal to do some form of portacaval shunt. However, best results of this procedure have been obtained in a younger age group. The esophageal varices persist following the shunting procedure, and in this instance control of persistent bleeding was of primary importance. Satisfactory palliation of hemorrhage can thus be accomplished according to Shafer.[17]

APLASTIC ANEMIA

Aplastic anemia is a term applied to a group of anemias characterized by a partial or total inhibition of bone marrow output, resulting in severe anemia, leukopenia, and thrombopenia, followed by a progressively fatal course in most patients. It seems advisable to consider aplastic anemia as a hematologic and clinical syndrome resulting from a variety of causes. The classification is confusing in the literature, but it seems logical that the term should be reserved for those cases in which there is an aplasia or hypoplasia of the bone marrow.

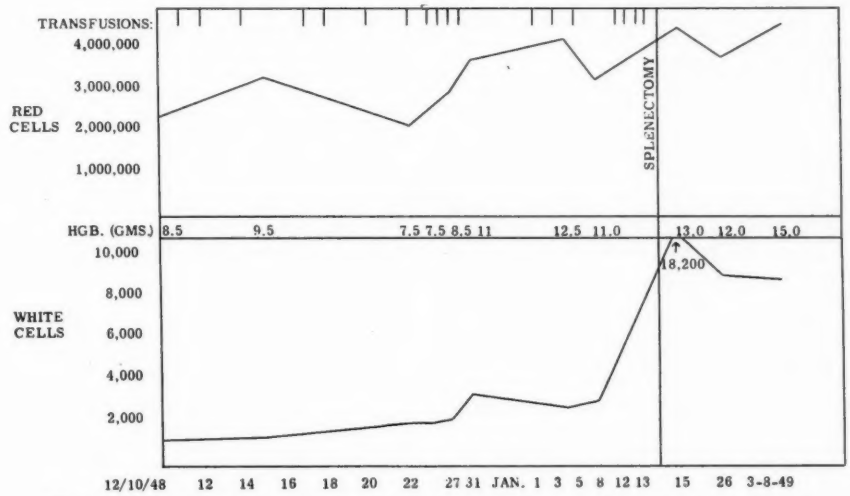

FIG. 2. The changing blood picture in a case of aplastic anemia (case 6) prior to and after splenectomy.

There is no characteristic age, race, sex, or familial distribution. Some cases are secondary to other disorders such as malignancy, tuberculosis, Hodgkin's disease, cirrhosis, nephritis, subacute bacterial endocarditis, leukemic infiltration, and benzene poisoning. Obviously, these would not be helped by splenectomy. However, in some cases of primary refractory and hypoplastic anemias, the operation is sometimes resorted to on the basis of removing the normal inhibitory effect of the spleen upon the bone marrow. Generally, in any case in which the bone marrow is hypoplastic or aplastic, the ultimate effect of removal of the spleen is, at best, in doubt. In certain cases of selected patients in whom no cause for the disease can be determined and who have been observed for a long period of time with no evidence of response

to medical therapy, splenectomy is warranted, even when the spleen is normal in size. This may result in restoration of a normal or a nearly normal blood picture. I was fortunate enough to see such a patient in case 6.

CASE 6.—Mrs. W. A. H., a 48 year old dietitian, a white woman, was admitted to the hospital December 10, 1948, with a history of a poor dietary intake for six months, with a weight loss of 20 pounds. She had had arthritic symptoms for several years. In August, 1948, she was found to have a leukopenia and later an accompanying anemia. There was no history of contact with known toxic substances, although she had been a self-medicator and had taken a variety of self-prescribed drugs.

Physical examination revealed a pale, sallow, middle-aged woman who appeared seriously ill. The pharynx was red with scattered hemorrhagic areas. The cervical nodes were moderately enlarged. The spleen was markedly enlarged, firm, and nontender.

On admission, the red blood cell count was 2,660,000; the hemoglobin was 55 per cent; the white blood cell count was 1,200 with 26 per cent neutrophils. The bone marrow was reported as being markedly aplastic for all cellular elements. After a period of five weeks, during which time the patient was given penicillin, Pentonucleotide, liver extract, and almost daily transfusions, the red blood cell count was 4,380,000; the hemoglobin, 81 per cent; and the white blood cell count was 2,900 with 36 per cent neutrophils. The patient remained acutely ill in appearance, was apprehensive and poorly cooperative, and would not eat. Splenectomy was done and two days thereafter the red blood cell count was 4,500,000; the hemoglobin was 84 per cent; and the white blood cell count was 19,750 with 80 per cent neutrophils. Figure 2 shows graphically the changes in the blood picture.

Over a subsequent four year observation period the patient has been clinically well. The peripheral blood cell count has been normal but with a low proportion of neutrophils.

Comment: The decision to perform a splenectomy on this patient was based simply on continuation of a severe illness without evidence of alleviation in spite of extravagant supportive measures and in the absence of known toxic, medicinal, or bacterial factors. Ordinarily the treatment of choice is supportive replacement of fresh citrated blood transfusions. Furthermore, the significance of splenomegaly should not be mistaken, since extramedullary hematopoiesis in liver and spleen may partially compensate for the marrow hypoplasia.[7]

SUMMARY AND CONCLUSION

A review of the literature has been made and a classification of the indications and contraindications for splenectomy presented. Six exemplary case histories are included for the purpose of discussion of the underlying splenic mechanism and establishment of the differential diagnosis.

It should be emphasized that a careful history as regards dietary habits, gastrointestinal complaints, bleeding, chemical exposure, hereditary disease and the like, coupled with a careful physical examination and an evaluation of the blood picture usually will lead to a fairly conclusive idea as to the possible etiologic mechanism. A few additional studies will ordinarily establish a final diagnosis. On this basis, the indications for or against splenectomy can be arranged in an orderly fashion, and if the operation is indicated, the results following splenectomy will prove beneficial to the patient.

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ABSTRACT OF DISCUSSION

DR. C. T. ASHWORTH, Fort Worth: The discussion given by Dr. Nitschke has been thorough and instructive and with the basic principles he has mentioned, I can agree.

Regarding the case of familial hemolytic anemia reported here, there are certain unusual features in the laboratory findings that require some comment. I refer to the absence of reticulocytosis, the normal range of stool urobilinogen, and the borderline erythrocyte fragility test. Also it is unusual to find the amount of icterus seen in this patient, 8.3 mg. of bilirubin per 100 cc. Still, in these cases of familial hemolytic anemia, we should be prepared to see a varying hematologic picture. In fact, one of the most notable contributions to the study of this disease recently is the finding of Owren that the first phase of the relapse in familial hemolytic anemia is that of marrow hypoplasia and the anemia is of the hypoplastic type. The hyperactive erythroid marrow with reticulocytosis seems to follow this stage shortly, and it is at this time that the patient usually presents himself for study. This aplastic stage of the disease should be kept in mind in following these cases to understand the varying hematologic picture.

I believe that the study of the bone marrow is an indispensable part of the evaluation of the cause, mechanism, and appropriate treatment of thrombocytopenic purpura. I cannot believe that splenectomy ever should be carried out in these cases until a fully satisfactory study of the marrow has been accomplished. If there is any criticism at all to be made of the case presented by Dr. Nitschke here, it might be the absence of a study of the bone marrow in the stage of the disease just before splenectomy. The purpose of bone marrow examination is first, to exclude aleukemic leukemia and aplastic anemia as the cause of the thrombocytopenia and second, to establish evidence favoring destruction of platelets by the spleen, consequently providing evidence which will indicate the value of splenectomy. This type of study requires careful morphologic evaluation including estimation of the number of megakaryocytes, a differential count of megakaryocytes according to age, and determination of the state of platelet production within the cytoplasm of the megakaryocytes.

In the case presented here, the spleen was enlarged and this phenomenon is somewhat unusual in cases of essential thrombocytopenic purpura. I would be interested to know what the pathologic study of the spleen revealed.

In the study of cases of splenic panhematopenia it is thought that the laboratory findings can be correlated with the clinical picture in such a way that, with little chance for error, the combination of findings can be considered diagnostic. Pancytopenia, or any combination of anemia, leukopenia, or thrombocytopenia, plus evidence of peripheral destruction especially of erythrocytes, plus a hyperactive marrow, plus splenomegaly indicates hypersplenism. The differentiation of the primary and secondary cases, however, is more difficult and may not be accomplished until the spleen has been removed and studied histologically. There seems to be difference of opinion about the relative value of the Adrenalin test in these cases. I have found it helpful frequently but agree with others that it cannot be interpreted exclusive of other data.

It is difficult to believe that any case of true aplastic anemia can be benefited by splenectomy. In the defense of this position I would submit the impression that aplastic anemia, though its diagnosis might seem simple, is one of the most commonly misdiagnosed of the severe anemias. The reason for this, I believe, is the faith that is often placed in a single bone marrow study. Failure to obtain marrow particles, dilution with sinusoidal blood, and the difficulty in aspiration of some solid, cellular lesions which may replace marrow are some of the causes for the low cell content that may be encountered in bone marrow smears and interpreted as aplastic anemia while other lesions are actually present. I believe that histologic sections of bone marrow particles which were aspirated or actual bone marrow biopsy specimens should always be available before deciding that a marrow is aplastic or hypoplastic.

1952 American Health Reviewed

The health of the American people in 1952 has been at a peak and the prospect is excellent for 1953, according to Dr. Louis I. Dublin, second vice-president and statistician of the Metropolitan Life Insurance Company.

The brightest spot in the year's record, believes Dr. Dublin, was the continued rapid fall in the mortality from tuberculosis. The death rate from the disease in 1952, he said, is likely to be less than 18 per 100,000, and noted that "it is difficult to realize that only as recently as 1946 it was double this rate. . . ."

Other statistical comments concerning the nation's health for 1952 given by Dr. Dublin include:

The infant mortality rate for 1952 is estimated at a little over 28 per 1,000 live births. The maternal mortality rate is estimated at less than 7 in every 10,000 live births.

The death rate in the United States was 9.6 per 1,000 in 1952, the fifth year in succession with a rate under 10.

A trend in respiratory disease was noted during 1952. The death rate from pneumonia and influenza was somewhat less than in 1951.

Communicable diseases of childhood caused relatively few deaths. Despite the fact that 1952 was a measles year, the death rate from the disease was low.

The exception to the generally favorable health situation in childhood was the high prevalence of poliomyelitis. More than 57,000 cases were reported during the year, the highest on record and actually one-third greater than the previous high of 1949.

In summarizing the outlook for 1953, Dr. Dublin stated, ". . . our major job in the health field in the year ahead is to press the fight vigorously on those diseases and conditions which are preventable or which claim most lives. . . ."

NEW INSULIN PREPARATIONS

Three new long-acting insulin preparations which offer promise in reducing the complexities of insulin administration to diabetics were described in the December 27 *Journal of the American Medical Association.*

Consisting of insulin mixed with small amounts of zinc, the new preparations have activity ranges from about eighteen to more than thirty hours, according to K. Hallas-Moller, Ph. D.; M. Jersild, M. D.; K. Petersen, M. Sc.; and J. Schlichtkrull, M. Sc., of Copenhagen, Denmark.

The authors stated that preliminary studies of 65 patients with severe cases of diabetes have shown "that satisfactory blood sugar control can be obtained in such patients with a single injection of the appropriate one of the three types of zinc insulin."

Zinc insulin was given to the patients daily at 8 a. m. The amounts of insulin varied according to individual reactions to the drug.

SUDDEN AND UNEXPECTED NATURAL DEATH

MERVIN H. GROSSMAN, M.D., Houston, Texas

IN 1951 there were more than 150 cases of sudden and unexpected death in the city of Houston, and in 101 cases an autopsy was ordered by the justice of the peace to determine the cause of death. These examinations were performed through the office of the county health officer, under the direction of Dr. W. W. Coulter, Sr. Perhaps it is better to emphasize the unexpected character of these deaths occurring in apparently healthy people rather than their suddenness. The term "natural death" implies that death was due entirely to disease and that trauma did not play any role in its causation.

It is difficult at times to draw distinct lines between natural death and death that may have been remotely initiated by some external factor. For example, fatty change of the liver may be a sequel to acute alcoholism, the former condition coming within the scope of this paper and the latter obviously excluded.

Death ordinarily comes after termination of an illness of longer or shorter duration, in which case the clinician has, or should have, some knowledge of the lesions concerned and of the organ involved. But, in some cases, death is either instantaneous or follows an illness of relatively short duration in a person whose previous health has been good. These cases are tragic for friends and relatives and often are puzzling to the physician, who sometimes lacks knowledge of the clinical history. In the absence of an autopsy, the signing of the death certificate presents no little difficulty, and as yet, only a fraction of the cases of sudden and unexpected death come to autopsy. Accuracy in assigning the cause of death often assumes considerable importance. There is a medicolegal aspect to cases of sudden and unexpected death, and every pathologist of experience has had occasion to perform autopsies on bodies exhumed because of persistent rumors of foul play.

Circumstances under which death occurred are important in deciding when postmortem examination is advisable. The presence of witnesses usually aids in confirming natural death so that autopsy is not requested whereas cases in which there are no witnesses usually make autopsies advisable.

From the Department of Pathology, Baylor University College of Medicine. Dr. Grossman is now at Memorial Hospital, Chattanooga, Tenn.

Read before the combined Sections on General Practice and Clinical Pathology, Texas Medical Association, Annual Session, Dallas, May 7, 1952.

Lesions responsible for sudden and unexpected natural death can be classified generally in three categories, Helpern and Rabson pointed out.[5] The largest group consists of natural disease processes which are slow in developing and damage a vital organ without producing any apparent symptoms. Sudden cessation of function results. Sudden and unexpected rupture of blood vessels with occurrence of fatal hemorrhage makes up the second category. Potential infectious diseases are in the third group.

"Of the three," Helpern and Rabson stated, "vascular rupture with hemorrhage is the easiest for the pathologist to evaluate as a necessary immediate cause of death. Infection is perhaps less obvious, whereas chronic degenerative disease or neoplasia is most difficult to assay. In this last category there is always the possibility of a more subtle, immediate, not necessarily natural cause of death which may be overlooked. For example, an autopsy may disclose a degree of coronary arteriosclerosis deemed sufficient to cause death. Chemical examination of the organs in such a case, however, may reveal a lethal amount of poison, or dissection of the vertebral column a broken neck with compression of the spinal cord. These chronic diseases are ordinarily of such slow progress that there is but little difference, if any, in their status ante mortem or at death. It is in these cases that a complete autopsy is most essential to exclude other conditions before ascribing death to the chronic disease process in question.[5]

Review of previously published literature and of the figures in Houston reflects an interesting anthropologic composition of the materials presented. One is struck by the paucity of women. Of 2,030 cases in a New York study,[5] only 19 per cent were women, although according to the 1940 census, 51 per cent of the inhabitants of Manhattan Island were female. In Houston in 1951, only 29 per cent of the cases were of females. When a racial analysis was made in the New York study, it was found that each race was represented among sudden and natural deaths in almost the same proportion as in the total population. In the same series, however, there was an exception in that Negro women, who are outnumbered by white women almost 5 to 1, made up more than 26 per cent of all female cases of sudden natural death, twice the expected proportion. It would seem that among the Negro women disease symp-

toms are not as apt to be heeded; medical care is sought less frequently so that death often takes place without a physician in attendance.

In a 1934 survey, Hamman,[4] who studied the analysis of a number of reports in the literature, concluded that 91 per cent of sudden death by natural causes are due to disease of the cardiovascular system; 65 per cent of all cases are due to heart failure; 21 per cent to hemorrhage; and 5 per cent to arterial embolism and thrombosis.

Deadman's figures[3] over the past twenty years, covering about 300 cases, were similar to Hamman's conclusions.

Moritz[7] noted that throughout the country figures indicate that between 10 per cent and 15 per cent of all deaths occur unexpectedly. The incidence is higher in urban areas than in rural areas. It is higher where a large proportion of the population is underprivileged or uneducated. Unexpected death occurs about twice as frequently in males as in females. It occurs at all ages. There are two peaks to its occurrence. Unexpected death occurs with particular frequency during the first year of life; then there is a gradual increase in frequency from the age of 35 on, unexpected deaths constituting about 16 per cent of all deaths between the ages of 35 and 65 years.

Table 1 is a comparison between the Houston series and several series previously published showing the incidence of causes of death.

TABLE 1.—*Site of Causes of Sudden and Unexpected Natural Deaths.*

Site of Lesion	Helpern[5] 2,030 Cases %	Weyrich[10] 2,668 Cases %	Lauren[9] 403 Cases %	Grossman 100 Cases %
Heart and aorta	45	42	51	37
Respiratory system	23	23	12	22
Brain and meninges	18	9	15	15
Digestive and urogenital tracts	10	13	9	1
Miscellaneous	4	13	12	13
Undetermined				12

INFANT DEATHS

Special mention should be made of why babies die unexpectedly. Potter[9] stated that the causes of disease and death during intrauterine life and in the first few weeks after birth are different in general from those appearing later in life. Diseases at all ages result from heredity or environment, or a combination of these factors. The younger the person, the greater is the role played by heredity; with the passage of each year, the greater the part played by environment.

The principal cause of death before birth and in the first few weeks of extrauterine life is interference with oxygenation. Premature separation of the placenta and prolapse of the cord, or entanglement of the cord, are the two most common factors involved. In the neonatal period prematurity is most responsible, especially in cases of babies weighing less than 1,000 Gm. The majority of deaths of infants under 1 year of age occur before the end of the first month, and after the first month pulmonary infection is the leading cause.

In premature babies weighing less than 2,000 Gm. intracranial hemorrhage is the leading cause of death. Also to be indicted are malformation, hemolytic disease of the newborn due to Rh incompatibility and within the A-B-O typing system, immaturity of the lungs, resorption atelectasis with hyaline-like membranes, and pneumonia.

Moritz[7] stated that "In 10 to 15 per cent of babies the autopsy fails to disclose any cause of death. Around 80 per cent of them die of unrecognized infections.... I did not realize until late in my medical experience that babies can be very sick but still eat and apparently act like normal babies. They can have a great deal of disease and still take a pretty full feeding. I have seen babies who were reported to take a full feeding at twelve o'clock and were dead at four with practically no area of lung tissue left, an interstitial exudative pneumonia that had simply expanded the interstitial tissue of the lung until there was no room left for air sacs."

What about status thymicolymphaticus? It is not the consensus of modern pathologists that status thymicolymphaticus is an existing entity because status thymicolymphaticus as a cause of death in childhood was a concept erected on a falsehood in the beginning. Moritz called attention to the problem thus: "Not until pathologists started examining the bodies of normal children, who died instantly of an automobile accident, was it realized that the normal thymus and normal lymphoid tissue was very abundant in the normal child. It used to be felt that, if a child at the age of three had a thymus that weighed fifty-five Gm., that was evidence of status thymolymphaticus. That was where the whole concept arose. I have never seen a death from it and I doubt that anyone else has."[7] As newer editions of the pathology texts state, status thymicolymphaticus is frowned upon more and more as a pathologic entity, and should soon come into the realm of nonexistence.

We still see newspaper articles concerning an infant found dead in its mother's bed, the death being ascribed to suffocation. Occasionally an autopsy report is seen in which the cause of death is deemed to be idiopathic suffocation. In light of newer knowledge, many of these deaths are found to be due to influenzal epiglottitis, Salmonella infection in the intestine, or meningitis with minimal or essentially no histologic aberrations. Cultures from the epiglottis, cecum, and cerebrospinal fluid are essential for a complete postmortem examination of an infant.

OBSCURE CAUSES

Moritz and Zamcheck[8] reviewed 1,000 cases of sudden deaths in apparently healthy, young soldiers between the ages of 18 and 40 during the period from 1942 to 1946. A review of 800 cases yielded five principal categories. The first was heart disease. Of 350 cases of heart disease, 300 deaths were due to coronary artery disease. In 91 cases of intracranial hemorrhage, the second category, 61 had ruptured aneurysms. The third category included 110 cases of meningococcemia, some associated with Waterhouse-Friderichsen syndrome. The fourth large group was miscellaneous. Of those cases in the miscellaneous group, respiratory disease contributed the largest number of deaths. There were other isolated causes such as adrenal atrophy, diabetes, hepatitis, pancreatitis, and septicemia. Obscure causes resulted in death in 140 cases making up the fifth group. Complete autopsies were done. Sections were prepared from every type of tissue and no cause of death was found. Cases were accepted as falling in this group only when the pathologist was competent and after all tissues had been examined and the records were so complete that injury or poison could be excluded. There were still 140 persons in whom there was not found one single change in the body which could be named as the plausible cause of death.

In 1897, the French medicolegal authority Brouardel[2] said, "Gentlemen, however carefully we may perform every autopsy, however minute our exploration of the body may be, however thorough may be our knowledge of the causes of sudden death, we sometimes meet with cases which it is impossible to explain. The proportion is about 8 or 10%." In the passage of time the opinion has changed. In 1947 Deadman[3] expressed the belief that with the present development of bacteriologic, biochemical, and toxicologic analysis and with the improvement of histologic technique and diagnosis, it would appear that the percentage of unsolved cases in the hands of competent pathologists with adequate laboratory facilities should be kept below 1 per cent. He pointed out, however, that there will always remain a few cases for which no solution can be found, so limited is our knowledge of the vital processes of life and of the conditions which fatally interfere with them.

CONCLUSION

The determination of the cause of death is basically an interpretive process. The latter includes recognition of the pathologic change, anatomically, chemically, and bacteriologically, and selection of that lesion or lesions which are necessarily fatal at the time when, and under the circumstances during which, death occurs. All of this presupposes a complete autopsy examination.

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ABSTRACT OF DISCUSSION

DR. J. M. TRAVIS. Jacksonville: I should like to encourage all general practitioners to obtain a reprint of Dr. Grossman's paper. Many of us do not have pathologists available, and many times with a pathologist available, it is not possible to obtain an authorization for a postmortem examination. We are presented with the problem of attempting to arrive at a reasonably correct diagnosis with only a meager history or no history available. Since Dr. Grossman has discussed the more common causes of unexpected death in each age group, we may run through the list and perhaps arrive at the probable diagnosis by selecting the diagnosis which seems to fit the case.

Many of our clinical diagnoses are based on knowing which diseases are apt to occur in certain age groups. For example, I recently had a 5 year old girl come into the hospital with a history of having recovered from mumps two weeks earlier. When I saw her, she had been ill about seven hours. She was still able to climb on the examining table. She was covered with petechiae and ecchymoses and had 102 F. temperature. Knowing that meningococcemia is one of the more common conditions to produce such a picture, we made smears from her petechiae and found Diplococci within the polymorphonuclear leukocytes. They were also present in many of the polymorphonuclear leukocytes obtained by venipuncture. Thus, we had the diagnosis within thirty minutes and also knew that her prognosis was poor. She died within twelve hours of the onset of her illness, and we were not able to obtain permission for a postmortem examination. Had we not immediately suspected meningococcemia, we would have had to guess at a diagnosis to put on the death certificate. I note that Dr. Grossman has mentioned meningococcemia as causing 110 deaths in a review of 800 cases of sudden death. That was in a group of soldiers, but of course, we know that children have the disease also.

Sometimes as practitioners we overlook the fact that we are obligated to ourselves and to our patients to make accurate diagnosis, even though it may be after a patient dies. Particularly since the advent of the sulfonamide drugs and the antibiotic agents, we find it easy to "shotgun" the patient with triple sulfa, penicillin, aureomycin, and streptomycin, without making a serious attempt to make a confirmed diagnosis. We console ourselves with the thought that the patient is more interested in getting well than in a diagnosis, but more and more patients are expecting correct

diagnoses. Even in those cases in which the patients "just want a quick cure," we can persuade most of them that an accurate diagnosis is important. The real problem is in convincing ourselves.

We should encourage the pathologist to do a complete autopsy. We can accomplish that in several ways. First is by being interested enough in a case to obtain permission for a postmortem examination. We can help by explaining to the family that it will be "complete," including an examination of the head. In my community there have been complaints from the embalmer and the family after a head was examined. Usually the proper conversation with the family will avoid this difficulty. A body which has been examined does require more time for embalming, but a good embalmer can handle it. We should never get permission for an examination of a single organ or a single cavity unless no other type of permission is available.

Get acquainted with the justice of the peace in your community. He can authorize an autopsy on any case in which you cannot make a diagnosis; however, he may not like to do it. You will have to convince him of the importance of an autopsy in a case where death was due to "natural causes." It is a Texas law that if a physician is in doubt about the cause of death, he can make application to the justice of the peace for authorization of an autopsy to determine the cause of death. The justice of the peace has the authority to order the autopsy.

MEDICAL PROBLEMS OF AN AGING POPULATION

EDWIN G. FABER, M.D., Tyler, Texas

No consideration of the medical problems of the old can be divorced from the social problems which are presented by this group. Furthermore, just as few diseases affect only the geriatric group, so the social problems of this class affect all age groups in society.

As a result of advances in medical knowledge and public health practice, life expectancy has increased from 48 years in 1900 to 67.2 years in 1948. This extension of the span of life has dislocated the usual age patterns of workers. Decreasing birth and death rates with marked immigration restrictions have reduced the number of young workers and increased the proportion of people in the older age groups. Although it is not within the scope of this paper to discuss the economic and social changes necessitated by this social shift, it is apparent that these factors directly affect the medical problems of the aged.

Geriatrics is a comparatively recent field of medical specialization, yet from time immemorial physicians in all fields of medicine have cared for the aged. There is no place to begin a discussion of their specific problems and no place to end. A full consideration of medicine and surgery with all their subspecialties would have to be included.

The attitude and approach of the physician to the older patient is of the greatest importance. The law considers us old and unfit for work at the arbitrary age of 65. With this general belief fostered by our government, the busy physician finds it easy to give the older patient little consideration when he complains of tiring easily or of general aches and pains that do not suggest grave disease. How many of us have listened to these complaints and thought, "Well, what does he expect at his age?" The younger the physician, the earlier old age sets in, in his patients. It is the rule rather than the exception that a half-hearted search for organic disease is made. Variations from the normal, if not too great, are attributed to old age and either disregarded or treated lightly. If organic disease is discovered, there is essentially no difference in the disease pattern in the young or old. Conditions such as malignancies, tuberculosis, heart and kidney disease require expert medical and surgical care and with minor changes present the same problems in all age groups.

The social aspect of the subject, however, presents special problems. Many old people are cross and querulous because they feel left out of the social and economic life of their community. As their resentment increases, they become careless of their personal appearance and their manners. If this continues, they become physically dirty and develop obnoxious habits that further isolate them from society and consequently increase their loneliness and resentment. In our present civilization old age is looked on as an affliction, and a great deal of effort is expended in schemes for the care of the aged. If we directed our plans to preventing the distressing appurtenances of old age, many of the problems now demanding attention would be solved and the economic value of skills practiced over many years would add to our productivity.

The trite expression of "growing old gracefully" needs our special thought, and the advice given the patient must be carefully considered. Much of the success of gracefully growing old depends on measures instituted before a person is old. The psychological adjustment to curtailment of physical and mental activities must start when the person is still active in his work. Hobbies must be acquired and the hobbyist urged to strive for as much skill as possible in his

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chosen avocation. One's pleasure in any sport or craft is in direct proportion to his skill.

There are some special conditions accompanying advanced age which predispose to psychogenic disorders of varying degrees. The most important are as follows: (1) concern over health, (2) worry over finances and fear of losing security, (3) loneliness and fear of not being wanted, (4) feeling of having outlived one's usefulness, and (5) fear of losing the place formerly enjoyed in the home and community. If the physician will consider these concerns of the aged patient, he will have a clearer insight into his many problems and be more able to evaluate the situation.

Since the care of the aged sick presents few special problems, our primary concern as physicians is to attempt to alleviate the mental distress of this group. When the aged seek relief from distressing symptoms, be they functional or organic, our solution of the problem depends entirely on the approach. Old people frequently resent advice from younger people. This should be kept in mind during the early part of the consultation or until a good rapport is established. Directions should be explicit and carefully explained. Since it is difficult to "teach old dogs new tricks," patience should be exercised. To reprimand harshly the aged patient for failure to follow instructions will frequently produce resentment and loss of confidence which may have taken months to establish. Insistence on doing everything the physician's way in minor matters may so irritate the patient that the whole program will fail. However, coddling the patient should not extend to the important measures advised, and strict adherence to the regimen ordered should be insisted on.

SPECIFIC MEASURES

I should like to mention a few specific measures which often appear important and arise commonly. The diet is frequently deficient, particularly in protein. Many old people eat little or no meat, and when it is recommended that they add this item to their diet, they complain that they not only cannot afford it but do not like it and cannot chew it. Adequate amounts of protein in some form must be provided. It is not uncommon to see peripheral edema on the basis of hypoproteinemia diagnosed as congestive failure in the aged cardiac patient. Treatment fails to correct the edema if the plasma protein is not restored. Careful dietary history will suggest other important deficiencies, since frequently the aged patient has a poor appetite. Because of the preoccupation of the aged with foods that they think disagree with them and cause constipation, they alter their diets in an attempt to relieve these fancied troubles. Rarely do I take a history on an old person that he does not tell me he has had trouble with constipation for years. Long years of belief that copious and frequent bowel movements are necessary to keep healthy has focused their attention on the bowel, and no amount of explanation can dispel this fallacy. Subclinical vitamin deficiency is probably more common in the aged than in any other segment of the general population. It seems that use of vitamins in this group is more justified than in any other except those that show definite clinical evidence of deficiency.

The aging process produces many physical changes which are invisible, but changes in appearance which are unsightly should be prevented whenever possible. The stooped posture can be prevented with proper exercises and deep breathing. Blemishes on the skin should be removed before they become large. Facial contours can be maintained with properly fitted dentures. These measures can be summed up in the general recommendations outlined by Seidel.[1] The aged person should:

1. Continue to work at his vocation and retain his accustomed position in society as long as possible.
2. Avoid sedentary habits and inactivity, continuing with whatever open-air activities or hobbies he may be accustomed to; if he has none, he should acquire some.
3. Engage in systematic daily exercises designed to prevent or overcome physical deformities; these should include the practice of deep breathing.
4. Make full use of helpful devices such as hearing aids, eyeglasses, and dentures.
5. Keep face and body free from unsightly blemishes.
6. Always maintain self-respect and dignity, be jealous of his appearance, dress neatly, and retain good manners.
7. Continue contact with lifetime friends, and learn to make new ones.
8. Follow good body hygiene and sanitation; eat sensibly—a well-balanced diet of nourishing food.
9. Seek medical advice at frequent intervals and carry out in full the advice given.
10. Above all, avoid despair and an outlook of futility, and "Fear God and keep his commandments; for this is all that is required of man." (Ecclesiastes XI:13)

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ABSTRACT OF DISCUSSION

DR. ERLE D. SELLERS, Abilene: As has been brought out in Dr. Faber's discussion, the problem of the aged patient has assumed increasing importance in recent years. The average person is living longer than was predicted even one or two decades ago. In a recent survey of our hospital patients, there was found a startlingly higher average age when compared with those of ten years back. Proper medical attention

for this group presents a real challenge to the medical profession.

I do not feel that geriatrics should be considered a specialty in itself, but more consideration must be given these patients by all physicians. Dr. Faber has brought to our attention some noteworthy approaches in handling these patients. Let me add that, although their medical care poses many particular problems, medical practice in general among these people often is not greatly different from that in younger age groups. Indeed, in retrospect, it is remarkable how well a majority of the aged respond to properly indicated medical and surgical treatment. I am not a surgeon, but I have been impressed in recent years how well even extremely elderly patients undergo major surgical procedures. The older concept that both elective and emergency needed surgery should be withheld with the excuse "he is too old to stand it" is no longer tenable.

In no other group is the doctor called on to practice the "art of medicine" as in this one. The physician has to treat the whole man, who often presents perplexing social and economic problems more difficult than the immediate medical situation.

Not the least of the problems in the medical care of the aged is the distressing mental deterioration that a majority of them present sooner or later. It is a doubtful service we have rendered our patient, his family, or friends if we have prolonged his life and suffering by various treatments, leaving his mind hopelessly clouded. It is difficult to prevent an attitude of despair in our own minds in such situations. All of us, however, have seen remarkable improvement in the mental faculties in some of these people when immediate physical ailments have been remedied. Certain medications, too, are at our disposal in improving cerebral circulatory disturbances and other derangements of the central nervous system. Anemias can be combated by proper treatment depending on their cause, and frequently gratifying improvement in the mental processes follows. The several newer antispasmodic drugs are sometimes of great help. Vasodilator drugs, particularly nicotinic acid, are worthy of trial. In my experience it is the most helpful drug in relieving the mental disturbances secondary to cerebral arteriosclerosis.

Dr. Faber has not been able in his paper to devote special attention to the early diagnosis and treatment of malignancy in these patients. An attitude of alertness may prove highly rewarding in this particular regard.

Another particular problem in practice among the aged is that increasing hospital costs and indeed lack of space render the general hospital impractical and inadequate for the prolonged attention many of these people need. One of the greatest needs in our medical set-up today is an increased number of hospital beds under a considerably different arrangement than our general hospitals, with their necessarily mechanized facilities and increased costs, afford. For the most part, the so-called rest homes, in our section of the state at least, are entirely inadequate and relegation of our friends and relatives to such places often approaches cruelty.

CASE REPORTS

Fibrocystic Disease of Pancreas and Lungs

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FIBROCYSTIC disease of the pancreas and lungs is a comparatively uncommon illness judged by the paucity of reports in the literature. In 1902 Bramwell attributed "infantilism" to pancreatic disease, and in 1913, Garrod and Hurtley reported 2 cases of pancreatic steatorrhea. However, it was not until 1938 that Andersen[1] described the first large series of cases showing a definite relationship between the intestinal and pulmonary manifestations and the pancreas.

ETIOLOGY AND PATHOLOGY

The etiology is unknown; however, the occurrence of fibrocystic pancreatic and pulmonary disease in siblings and in infancy suggests that there is a familial and congenital factor. In reported cases[1, 2] the history of two or three siblings dying before 1 year of age with the history of diarrhea, foul smelling stools, malnutrition, and recurrent lung infections is common. Undoubtedly some of these deaths were due to pancreatic disease. The pathologic findings also indicate a congenital development.

As the name implies, the pathologic picture is one of fibrosis and cyst formation within the pancreas and lungs. The primary lesion seemingly is an abnormality in the pancreatic enzymes resulting in precipitation of the secretions in the lumen of the ducts.[3] As the precipitations increase in size, the proximal ducts and acini dilate with flattening of the epithelium and cyst formation. Connective tissue is formed around the atrophic acini and cysts. When the enzymes are deficient during intrauterine development, the meconium is gray and hard in consistency and may produce intestinal obstruction by accumulation in the ileum. At birth the bronchial mucosa is nor-

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mal, but with the development of pancreatic deficiency, pulmonary changes occur. These changes are metaplasia of bronchial epithelium with loss of cilia and plugging of the bronchial tree resulting in bronchiectasis, pneumonia, pulmonary abscesses, atelectasis, cyst formation, and fibrosis. The mucosal changes in the lung have been attributed to vitamin A defiency resulting from inadequate intestinal absorption because of the pancreatic enzyme deficiency; however, there is some evidence to suggest a systemic involvement of the bronchi and pancreas with their secretory derangements resulting from a common origin.[3]

CLINICAL FINDINGS AND DIAGNOSIS

The clinical picture varies with the onset of the disease. When disease is present at birth, the infant usually dies soon after birth of intestinal obstruction due to meconium. When the illness develops after the first few weeks of life, the symptoms are related to the intestinal tract or respiratory system or both. The intestinal symptoms are the passage of frequent large stools which usually are greasy and foul smelling. There is failure to gain weight and weakness in the presence of a good appetite. The respiratory phase is characterized by repeated "colds" and attacks of "bronchitis" with frequent development of bron-

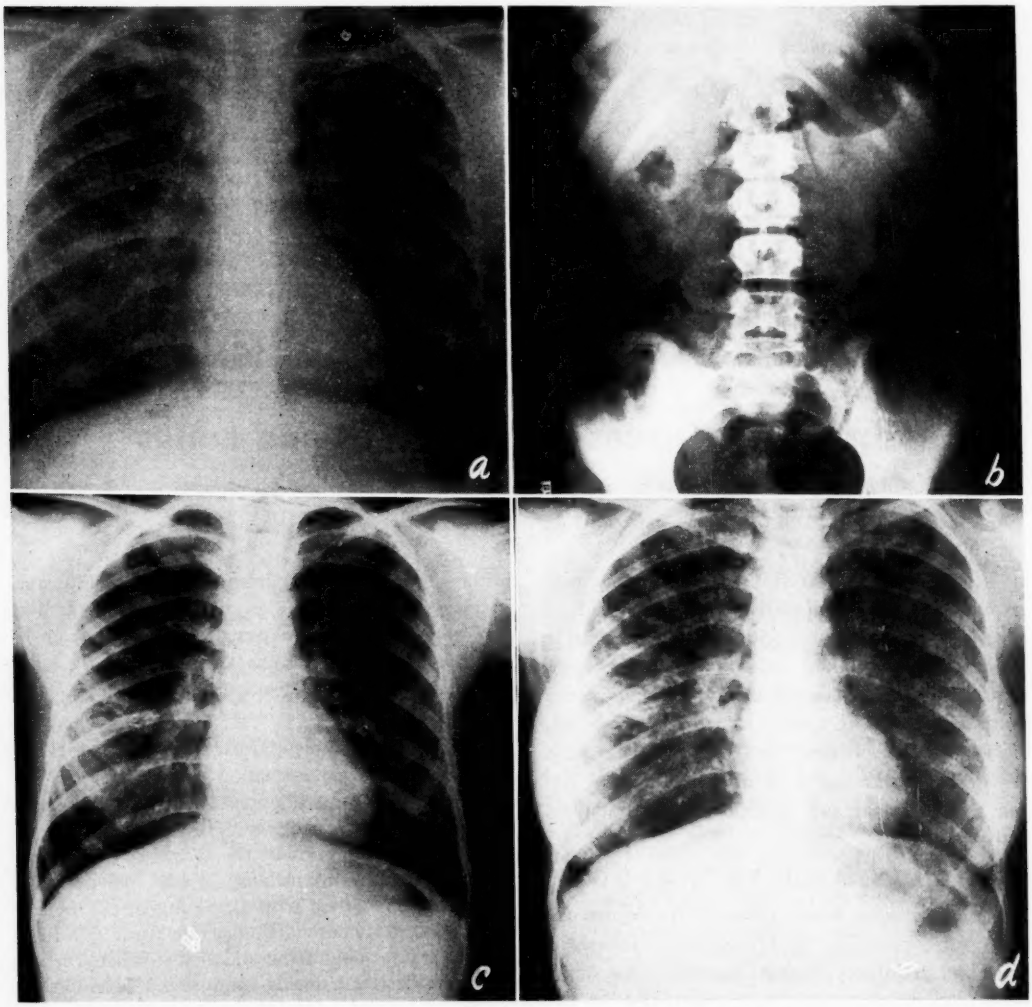

FIG. 1a. Roentgenogram taken early in the course of the disease showing bilateral pulmonary infiltrations.

b. Roentgenogram of the abdomen showing calcification in the region of the head of the pancreas.

c. Roentgenogram made after four months of therapy showing considerable clearing of the infiltrative process.

d. Roentgenogram taken after four years of therapy showing an increase in pulmonary infiltrations.

chopneumonia, pulmonary abscesses, and bronchiectasis. Death is usually due to respiratory rather than to intestinal involvement. Occasionally a patient is seen with chiefly respiratory findings, but the usual case presents both.

Laboratory aids in the diagnosis are (1) vitamin A absorption test, (2) secretin test, (3) glucose tolerance test, and (4) test for fat content of stools with determination of the percentage of free fat. The secretin test is of limited clinical value because of the technical difficulties, and there is some question as to the reliability of the free fatty acid to total fat. In pancreatic steatorrhea, free fatty acid content is low but Andersen[1] reported a patient with high percentage of split fat in the stools who at autopsy had cystic fibrosis of the pancreas.

The confusion resulting from stool fat studies has been due to failure to place the patient on a diet with a known amount of fat during the collection period and due to the fact that neutral fats in the stool continue to be split by the bacteria present even outside the body.

TREATMENT

Therapy is directed toward the pancreatic deficiency and pulmonary complications. Pancreatic extracts given before meals consisting of high carbohydrate, high protein, and low fat foods, and large doses of vitamin A are recommended. The pulmonary complications are most important and are managed best by frequent chemotherapy and the use of antibiotic drugs.

CASE REPORT

A. L., a white girl aged 13, was first seen by one of us (C. D. R.) in 1947 with the history of intestinal and pulmonary complaints since babyhood. When a baby, she was taken off breast milk because of frequent bulky, greasy stools, and when she was 2 years of age, celiac disease was diagnosed. Cough and "chest colds" always had been a problem, and when initially seen, she had been under observation for pulmonary tuberculosis fifteen months and received streptomycin for four months. Physical examination revealed a thin, chronically ill appearing girl 5 feet tall, weighing 76 pounds, and with temperature of 99.2 F. Scattered coarse inspiratory rales were heard over the lung fields. The liver edge was palpable at the right costal margin and the spleen was not palpable. There were no other significant findings. Stool specimens were not studied for quantitative fats, but on routine microscopic examination, the technician reported the stools as being too oily for parasite observation. Hemoglobin was 8.2 Gm.; erythrocytes 4,400,000 per cubic millimeter; leukocytes 8,400 with 74 per cent polymorphonuclears, 22 per cent lymphs, 3 per cent monocytes, and 1 per cent eosinophils. A chest roentgenogram (fig. 1a) showed bilateral pulmonary infiltration of both upper lung fields. Roentgenograms of the abdomen (fig. 1b) showed pancreatic calcification. The patient was placed on a regimen of oral pancreatic extracts, vitamins A and D, and penicillin, and within twenty-four hours the diarrhea stopped. There was rapid symptomatic improvement, and after two weeks of therapy she had gained 5 pounds and the lungs were clear to auscultation. A chest film taken after four months of therapy (fig. 1c) showed rather striking clearing of the areas of pulmonary infiltration. Pancreatic extract and vitamins A and D have been continued, and the patient has done well except for occasional chest infections which have responded to antibiotic therapy. She has one or two bowel movements of normal character daily. Radiologically there appears to have been an increase in the infiltration process (fig. 1d), and minimal clubbing of fingers is present. When last seen April 4, 1952, she weighed 110 pounds and was asymptomatic. She is now 18 years old and is an honor student in the eleventh grade of high school.

SUMMARY

Fibrocystic disease of the pancreas and lungs has been discussed briefly and a case occurring in an 18 year old girl has been presented. The diagnosis was established by the presence of typical symptoms, characteristic pancreatic and pulmonary roentgenographic changes, and a striking response to specific therapy. The unusual feature of this case is the apparent well-being of the patient with an illness which often is fatal in childhood. The exact benefit derived from a replacement therapy or antibiotic therapy cannot be definitely determined, but both probably were important in producing this gratifying response. The prognosis in this case can be determined only by further observation.

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ABSTRACT OF DISCUSSION

DR. DANIEL E. JENKINS, Houston: This review and case report brings to light a disease entity that has long been recognized by pediatricians, but largely neglected by internists. In fact, some of our most modern texts of medicine do not make mention of fibrocystic disease of the pancreas.

There are several important reasons why internists should be aware of fibrocystic disease of the pancreas. First, today many patients are surviving as a result of early diagnosis and prompt treatment with pancreatic replacement therapy and control of the pulmonary manifestations with antibiotic drugs. Consequently, more of these patients will be reaching adult life. Second, pulmonary emphysema is one of the most common incapacitating lung ailments known to the internist and often its etiology is obscure. I know of no study in which adult patients with pulmonary fibrosis or emphysema or both have been routinely surveyed for any evidence of pancreatic insufficiency. It is partly through an increased awareness by the pediatrician and the radiologist that diffuse pulmonary fibrosis and emphysema in infancy and childhood is commonly associated with fibrocystic disease of the pancreas that recognition and early diagnosis of the pancreatic disorder has been successful.

As it is the pulmonary component of this disease which

is largely responsible for death in those patients who survive the first few weeks of neonatal life, intensive therapy directed toward control of the progressive bronchial and pulmonary infection with its sequelae of atelectasis, emphysema, fibrosis, and bronchiectasis is of paramount importance. Andersen, in a rather impressive series of cases diagnosed prior to the onset of pulmonary complications, has shown that prompt pancreatic replacement therapy, maintenance of adequate nutrition, and prompt control of respiratory infection have been successful in avoiding entirely the pulmonary complications in most of the patients so managed.

NEWS

COMING MEETINGS AND CLINICS

Texas Medical Association, Houston, April 28-29, 1953. Dr. T. C. Terrell, Fort Worth, Pres.; Mr. N. C. Forrester, 1801 Lamar Blvd., Austin, Executive Secy.

American Medical Association, New York, June 1-5, 1953. Dr. Louis H. Bauer, Hempstead, N. Y., Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy. Dr. Walter S. Burrage, Milwaukee, Pres.; Mr. James O. Kelley, 208 E. Wisconsin Ave., Milwaukee 2, Executive Secy.

American Academy of Dermatology and Syphilology. Dr. C. Guy Lane, Boston, Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N. W., Cleveland 15, Secy.

American Academy of General Practice, St. Louis, March 23-26, 1953. Dr. R. B. Robins, Camden, Ark., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Obstetrics and Gynecology, Cincinnati, Dec. 14-16, 1953. Dr. Carl P. Huber, Indianapolis, Pres.; Dr. Ralph A. Reis, 116 S. Michigan Ave., Chicago 3, Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, 1953. Dr. Frederick C. Cordes, San Francisco, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Miami, Oct. 6-9, 1953. Dr. Phillip Barba, Philadelphia, Pres.; Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, San Francisco, March 27-30, 1953. Dr. Robert M. Janes, Toronto, Canada, Pres.; Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secy.

American Association of Genito-Urinary Surgeons, Hot Springs, Va., May 6-8, 1953. Dr. A. J. Scholl, Los Angeles, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., Sept. 10-12, 1953. Dr. Nicholson J. Eastman, Baltimore, Pres.; Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Exec. Vice-Pres.

American College of Allergists, Dr. J. Warrick Thomas, Richmond, Va., Pres.; Dr. Fred W. Wittich, 423 La Salle Medical Bldg., Minneapolis 2, Secy.

American College of Chest Physicians, New York, May 28-31, 1953. Dr. Andrew L. Banyai, Milwaukee, Pres.; Mr. Murray Kornfield, 112 E. Chestnut St., Chicago 11, Exec. Secy.

American College of Physicians, Atlantic City, April 13-17, 1953. Dr. T. Grier Miller, Philadelphia, Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Chicago, February, 1953. Dr. John S. Bouslog, Denver, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, Chicago, Oct. 5-9, 1953. Dr. Harold L. Foss, Danville, Pa., Pres.; Dr. Michael L. Mason, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine, Chicago, August, 1953. Dr. Walter M. Solomon, Cleveland, Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Dermatological Association, Lake Placid, N. Y., June 9-13, 1953. Dr. Earl D. Osborne, Buffalo, Pres.; Dr. L. A. Brunsting, 102 2nd Ave., S. W., Rochester, Minn., Secy.

American Gastro-Enterological Association, Atlantic City, May 1-2, 1953. Dr. Albert M. Snell, Palo Alto, Calif., Pres.; Dr. H. Marvin Pollard, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Gynecological Society. Dr. William P. Healy, New York, Pres.; Dr. John I. Brewer, 104 S. Michigan Ave., Chicago, Secy.

American Hospital Association. Dr. Anthony J. J. Rourke, San Francisco, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, New Orleans, April 28-30, 1953. Dr. Francis E. Le Jeune, New Orleans, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, June 15-17, 1953. Dr. Hans H. Reese, Madison, Wis., Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, Hot Springs, Va., May 28-30, 1953. Dr. Conrad Berens, New York, Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association. Dr. James Dickson, Cleveland, Pres.; Dr. George O. Eaton, 4 E. Madison St., Baltimore 2, Secy.

American Pediatric Society, Atlantic City, May 6-8, 1953. Dr. Irvine McQuarrie, Minneapolis, Pres.; Dr. Aims McGuinness, 237 Medical Laboratory, University of Pennsylvania, Philadelphia 46, Secy.

American Proctologic Society, Boston, June 10-13, 1953. Dr. Newton D. Smith, Rochester, Minn., Pres.; Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, N. Y., Secy.

American Psychiatric Association, Los Angeles, May 4-9, 1953. Dr. D. Ewen Cameron, Montreal, Canada, Pres.; Dr. R. Finley Gayle, 6300 Three Chopt Rd., Richmond 21, Va., Secy.

American Public Health Association. Dr. Gaylord W. Anderson, Minneapolis, Pres.; Dr. R. W. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists. Dr. Ralph T. Knight, Minneapolis, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists. Dr. Israel Davidsohn, Chicago, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Los Angeles, April 1-3, 1953. Dr. Robert S. Dinsmore, Cleveland, Pres.; Dr. Nathan Womack, Chapel Hill, N. C., Secy.

American Urological Association, St. Louis, May 11-14, 1953. Dr. George F. Cahill, New York, Pres.; Dr. C. H. DeT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Chicago, April 10-11, 1953. Dr. Charles L. Farrell, Pawtucket, R. I., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter. Tri-State Assembly, Dallas, Feb. 5-7, 1953. Dr. H. W. Meyerding, Rochester, Minn., Pres.; Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Los Angeles, May 18-22, 1953. Dr. Sidney J. Shipman, San Francisco, Pres.; Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secy.

Radiological Society of North America. Dr. Joseph C. Bell, Louisville, Ky., Pres.; Dr. D. S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, Atlanta, Ga., Oct. 26-29, 1953. Dr Walter C. Jones, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham 3, Ala., Secy.

Southern Psychiatric Association. Dr. R. Burke Suitt, Durham, N. C., Pres.; Dr. Newdigate M. Owensby, Medical Arts Bldg., Atlanta, Ga., Secy.

Southern Surgical Association. Dr. I. Mims Gage, New Orleans, Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville 5, Tenn., Secy.

Southwest Allergy Forum, Kansas City, Mo., 1953. Dr. Orval R. Withers, Kansas City, Mo., Pres.; Dr. James Holman, Medical Arts Bldg., Dallas, Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association. Dr. James Walsh, Douglas, Ariz., Pres.; Dr. W. W. Schuessler, 1415 First National Bank Bldg., El Paso, Secy.

Southwestern Surgical Congress. Dr. Michael E. DeBakey, Houston, Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.
Tri-State Medical Assembly. Dr. Frank G. Thibault, El Dorado, Ark., Pres.; Dr. Albert M. Hand, Le Bonheur Children's Hospital, Memphis 3, Tenn., Secy.
United States-Mexico Border Public Health Association, Ciudad Juarez, Chihuahua, Mexico, 1953. Dr. Salvador Molina Velez, Nuevo Leon, Mexico, Pres.; Dr. Sidney B. Clark, 314 U. S. Court House, El Paso, Secy.

STATE

Private Clinics and Hospitals Association of Texas. Dr. S. D. Coleman, Navasota, Pres.; Dr. Neil D. Buie, Marlin, Secy.
State Tumor Clinic, Wichita Falls, April 1, 1953. Dr. Bailey R. Collins, 925½ Scott Street, Wichita Falls, Director.
Texas Academy of General Practice, Dallas, Sept. 21-23, 1953. Dr. C. U. Callan, Rotan, Pres.; Dr. Woodson W. Harris, 1410 Nickerson, Austin, Secy.
Texas Academy of Internal Medicine. Dr. W. C. Dine, Amarillo, Pres.; Dr. John S. Chapman, 3810 Swiss Ave., Dallas, Secy. Meetings restricted to members.
Texas Air-Medics Association, Houston, April 27, 1953. Dr. Claire F. Miller, Waco, Pres.; Dr. J. S. Minnett, 2512 Welborn, Dallas, Secy.
Texas Association of Blood Banks. Dr. W. N. Powell, Temple, Pres.; Miss Marjorie Saunders, 3500 Gaston Avenue, Dallas, Secy.
Texas Association of Obstetricians and Gynecologists, Fort Worth, Feb. 13-14, 1953. Dr. George Adam, Houston, Pres.; Dr. Carey Hiett, 815 Fifth Ave., Fort Worth, Secy.
Texas Chapter, American College of Chest Physicians, Houston, April 27, 1953. Dr. Robert B. Morrison, Austin, Pres.; Dr. Samuel Topperman, East Texas State Sanatorium, Tyler, Secy.
Texas Club of Internists. Dr. Herbert Hill, San Antonio, Pres.; Dr. Hatch W. Cummings, Jr., 203 Hermann Professional Bldg., Houston, Secy.
Texas Dermatological Society, Houston, April 26-27, 1953. Dr. C. H. McCuistion, Austin, Pres.; Dr. Thomas L. Shields, 1216 Pennsylvania Ave., Fort Worth, Secy.
Texas Diabetes Association, Houston, April 26, 1953. Dr. E. K. Doak, Houston, Pres.; Dr. Ivan Mayfield, 1318 Main, Lubbock, Secy.
Texas Division, American Cancer Society, Austin, Jan. 16-17, 1953. Mr. Frank C. Smith, Houston, Pres.; Mr. J. Louis Neff, 1609 Colorado, Austin, Executive Director.
Texas Heart Association, Houston, April 27, 1953. Dr. J. C. Crager, Beaumont, Pres.; Miss Roberta Miller, 411 Reserve Loan Life Bldg., Dallas, Executive Secy.
Texas Hospital Association, Galveston, May 12-14, 1953. Mr. Carroll H. McCray, Tyler, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.
Texas Neuropsychiatric Association, Houston, April 27, 1953. Dr. Don Morris, Dallas, Pres.; Dr. John L. Otto, John Sealy Hospital, Galveston, Secy.
Texas Orthopedic Association, Houston, April 27, 1953. Dr. Felix Butte, Dallas, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.
Texas Pediatric Society, Corpus Christi, October, 1953. Dr. J. A. Bybee, Beaumont, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.
Texas Public Health Association, Galveston, Feb. 15-18, 1953. Mr. H. E. Drumwright, Dallas, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.
Texas Radiological Society, San Antonio, Jan. 23-24, 1953. Dr. Robert D. Moreton, Fort Worth, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.
Texas Railway and Traumatic Surgical Association, Houston, April 27, 1953. Dr. Everett Lewis, Houston, Pres.; Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.
Texas Rheumatism Association. Dr. Robert H. Mitchell, Fort Worth, Pres.; Dr. Charles H. Cornwell, Marlin, Secy.
Texas Society for Mental Health, Austin, March 2-3, 1953. Mr. William Grant, Jr., Baytown, Pres.; Mrs. Elizabeth F. Gardner, 2504 Jarratt Ave., Austin 21, Executive Secy.
Texas Society of Anesthesiologists, Houston, April 27, 1953. Dr. John F. Winter, San Antonio, Pres.; Dr. C. R. Allen, John Sealy Hospital, Galveston, Secy.
Texas Society of Gastroenterologists and Proctologists, Houston, April 27, 1953. Dr. Jack Kerr, Dallas, Pres.; Dr. C. P. Hardwicke, 120 W. Seventh St., Austin, Secy.
Texas Society of Ophthalmology and Otolaryngology. Dr. J. Charles Dickson, Houston, Pres.; Dr. Lyle Hooker, 627 Esperson Bldg., Houston, Secy.
Texas Society of Pathologists, Houston, Jan. 25, 1953. Dr. Charles T. Ashworth, Fort Worth, Pres.; Dr. Lloyd R. Hershberger, Shannon Memorial Hospital, San Angelo, Secy.
Texas Surgical Society, San Antonio, April 6-7, 1953. Dr. George W. Waldron, Houston, Pres.; Dr. Albert W. Hartman, 414 Navarro St., San Antonio 5, Secy.
Texas Tuberculosis Association, Mineral Wells, April 9-11, 1953. Dr. David McCullough, Kerrville, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.
Texas Urological Society. Dr. R. E. Cone, Galveston, Pres.; Dr. Roy M. Morgan, Jr., Hermann Hospital, Houston, Secy.

DISTRICT

First District Society, El Paso, Feb. 13-14, 1953. Dr. Jim Camp, Pecos, Pres.; Dr. H. D. Garrett, First National Bldg., El Paso, Secy.
Second District Society, Big Spring, April 3, 1953. Dr. John Hogan, Big Spring, Pres.; Dr. Arch Carson, Box 111, Big Spring, Secy.
Third District Society, Lubbock, April 14-15, 1953. Dr. James A. Odom, Memphis, Pres.; Dr. James T. Hall, 1626 Fifteenth Street, Lubbock, Secy.
Fourth District Society, Brownwood, Fall, 1953. Dr. Lloyd R. Hershberger, San Angelo, Pres.; Dr. Paul M. Wheelis, Brownwood, Secy.
Fifth and Sixth Districts Society, Corpus Christi, July 10-11, 1953. Dr. Y. C. Smith, Sr., Corpus Christi, Pres.; Dr. Robert J. Sigler, 1126 Third Street, Corpus Christi, Secy.
Seventh District Society, Austin. Dr. George W. Tipton, Austin, Pres.; Dr. D. B. Faubion, 1403-C Guadalupe, Austin, Secy.
Eighth District Society, Fall, 1953. Dr. Andrew S. Tomb, Victoria, Pres.; Dr. George E. Glover, Jr., Victoria, Secy.
Ninth District Society, Baytown, April 1, 1953. Dr. Otto F. Schoenvogel, Brenham, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.
Tenth District Medical Society. Dr. Peyton C. Clements, Lufkin, Pres.; Dr. Edmund D. Jones, 2575 South, Beaumont, Secy.
Eleventh District Society, Tyler, Spring, 1953. Dr. Roscoe Moore, Mineola, Pres.; Dr. Marlin T. Braswell, Henderson, Secy.
Twelfth District Society. Dr. J. B. Brown, Temple, Pres.; Dr. Neil D. Buie, Marlin, Secy.
Thirteenth District Society, Wichita Falls. Dr. R. E. Cowan, Graham, Pres.; Dr. Robert D. Moreton, 815 Medical Arts Bldg., Fort Worth, Secy.
Fourteenth District Society, Gainesville, June 9, 1953. Dr. W. P. Philips, Greenville, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.
Fifteenth District Society, Daingerfield, 1953. Dr. D. R. Baber, Daingerfield, Pres.; Dr. P. A. Reitz, Pittsburg, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 16-19, 1953. Miss Betty Elmer, Medical Arts Bldg., Dallas 1, Executive Secy.
Central Texas Spring Clinic, Waco. Dr. Walter B. King, 2320 Columbus Ave., Waco, Secy.
International Medical Assembly of Southwest Texas, San Antonio, Jan. 27-29, 1953. Dr. John M. Smith, Jr., 205 Camden St., San Antonio, Secy.
New Orleans Graduate Medical Assembly, March 2-5, 1953. Dr. Woodard D. Beacham, Room 103, 1430 Tulane Ave., New Orleans 12, Secy.
North Texas-Southern Oklahoma Fall Clinical Conference, Sept. 16, 1953. Dr. C. H. Wilson, Wichita Falls, Chairman.
Oklahoma City Clinical Society Conference. Mrs. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.
Postgraduate Medical Assembly of South Texas, Houston, July 20-22, 1953. Dr. C. A. Dwyer, Secy., 229 Medical Arts Bldg., Houston.

Texas Neuropsychiatric Association

The regular fall meeting of the Texas Neuropsychiatric Association was held at the Veterans Administration Hospital in Houston, November 23, with Dr. Don P. Morris, Dallas, president, conducting the business and scientific programs.

The following scientific program prepared by Dr. William S. Fields was presented:

The Behavior and Unconscious Fantasies of Patients with Rheumatoid Arthritis—Sidney E. Cleveland, Ph. D., and Seymour Fisher, Ph. D., Psychology Section, Veterans Administration Hospital, Houston.
The Use of Mebaral in Alcoholism and Anxiety—Drs. Jackson A. Smith and R. Milton Finney, Department of Psychiatry and Neurology, Baylor University College of Medicine, Houston.
Cortical Representation of Cognitive Functions—David B. Vinson, Ph. D., Department of Psychiatry and Neurology, Baylor University College of Medicine, Houston.
Presentation of Cases—Staff, Veterans Administration Hospital, Houston.

Approved for membership in the association were Drs. Sam Hoerster, Big Spring; William C. Adamson and A. P. Rousos, Austin; Howard C. Reid and Victor J. Ellis, Amarillo; Harry M. Little, William S. Fields, Charles Gaitz, Isham Kimball, Jr., David Mendell, and Moses Ashkenazy,

Houston; and Michael J. Pescor and William F. Ossenfort, Dallas.

PERSONALS

Dr. J. Shirley Sweeney, Gainesville, spoke at the recent annual meeting of the Oklahoma Public Health Association, which was held at the University of Oklahoma, Norman. The subject of his address was "The Answer to Diabetes in Oklahoma."

Dr. John H. Vaughan, Amarillo, was one of the speakers at the fourth Inter-American Congress of Radiology, held in Mexico City from November 2 through 8.

Dr. Griff T. Ross, Mount Enterprise, and his family were guests of honor December 4 at an "Appreciation Night" staged in their behalf by the people of that community, according to news stories in the Mount Enterprise *Progress.*

Recent births reported by the *Alumni Bulletin* of the University of Texas Medical Branch include a boy to Dr. and Mrs. John W. Middleton, Galveston. The *Newsletter* of the Tarrant County Auxiliary also reports recent births, including boys to *Dr. and Mrs. Edgar L. Etier, Jr.* and to *Dr. and Mrs. Charles H. Riddle,* and a girl to *Dr. and Mrs. John Sewell,* all of Fort Worth.

Reeves Delegate to Worldwide Meeting

Dr. L. H. Reeves of Fort Worth, former president of the Texas Medical Association, has been named by Governor Allan Shivers to represent Texas at the joint meeting of the World Medical Association and the Pan American Medical Confederation to be held in Richmond, Va., April 23-25. The conference will be the first to be held by the W.M.A. in the western hemisphere, the seventy-fifth for the Pan American organization.

The Hon. John S. Battle, governor of Virginia, requested each of the other forty-seven governors to appoint a 75 year old physician from the state to attend the meeting and help celebrate the medical advances made within his lifetime.

Dr. Reeves, long active in the Tarrant County Medical Society, was president of the state medical organization in 1939-1940. For most of his professional career he has been active in medical organizations, having served many terms of office in various capacities both on local and state levels. At present he is chairman of the Committee on Memorial Exercises and a member of the Committee on Medical History and the Committee to Write a History of the Texas Medical Association.

OBSTETRICIANS AND GYNECOLOGISTS TO MEET

Dr. Christopher J. Duncan, associate professor of gynecology at Harvard University Medical School, Boston, will be guest speaker at the Texas Association of Obstetricians and Gynecologists' meeting February 13 and 14 at the Hilton Hotel, Fort Worth. Dr. Duncan, who is also associate chief surgeon at the Free Hospital for Women in Brookline, Mass., will speak on "The Ovarian Salvage at the Time of Routine Total Hysterectomy" and "Complications in Six Years of Routine Total Hysterectomy at the Free Hospital for Women."

The remainder of the program will be presented by members and local guests. Anyone interested in obstetrics and gynecology is invited to attend the scientific programs. A cocktail party and banquet for members, guests, and wives has been planned.

LIBRARY SECTION

SURGEON-GENERAL'S CATALOGUE WANTED

The Library of the Texas Medical Association is desirous of obtaining the following volumes of the Index Catalogue of the Library of the Surgeon-General's Office:

Second Series, Vol. 1, 2, 4, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, and 21.

Third Series, Vol. 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, and 21.

If any physician or group have in their possession any of these volumes and would care to make a gift of them to the Library, they will be gratefully accepted.

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Request for packages should be addressed "Library, Texas Medical Association, 1801 Lamar Boulevard, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the Library during December:

Reprints received, 971.
Journals received, 283.
Books received, 16.

Office Psychiatry, Moench; *Year Book of Pediatrics, 1952,* Gellis (editor); *Dermatology, Essentials of Diagnosis and Treatment,* Sulzberger and Wolf, Year Book Publishers, Chicago.

Textbook of Surgery, Moseley (editor); *Diseases of the Heart and Arteries,* Herrmann, C. V. Mosby, St. Louis.

Introduction to Physiological Optics, von Tschermak-Seysenegg; *Practical Blood Grouping Methods,* Wall; *B-Vitamins for Blood Formation,* Jukes; *The Pharmacology of Anesthetic Drugs,* Adriani; *The Biochemistry of Gastric Acid Secretion,* Conway; *Body Temperature, Its Changes with Environment, Disease and Therapy,* Selle, Charles C. Thomas, Springfield, Ill.

A Catalogue of Incunabula and Manuscripts in the Army Medical Library, Schullian and Sommer, Henry Schuman, Inc., New York.

Operating Room Technic, St. Mary's Hospital, Rochester, Minn., W. B. Saunders, Philadelphia.

The Principles and Practice of Medicine, Davidson, Livingstone Ltd., Edinburgh and London.

Biochemistry of Disease, ed. 2, Bodansky and Bodansky, MacMillan Co., New York.

The Literature on Streptomycin, 1944-1952, Waksman, Rutgers University Press, New Brunswick, N. J.

SUMMARY OF SERVICE

Local users, 81.
Local packages, 19.
Borrowers by mail, 69.
Packages mailed, 95.
Films loaned, 69.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be required to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films in the hands of the borrower.

Request for films should be addressed to "Motion Picture Film Library, Texas Medical Association, 1801 Lamar Boulevard, Austin, Texas." A list of available films, with descriptions, will be furnished on request.

The following films were loaned by the Library during December:

Accent on Use (National Foundation for Infantile Paralysis)—Gonzales High School, Gonzales.

A.M.A. Newsreel (American Medical Association)—Dr. R. W. Loveless, Bastrop.

Anesthesia, Low Spinal (Modified Saddle Block) in Obstetrics (Ciba Pharmaceutical Products)—Arlington State College, Arlington, and Kimble-Mason-Menard-McCulloch Counties Medical Society, Brady.

Another to Conquer (Texas Tuberculosis Association)—Gonzales High School, Gonzales.

Ascorbic Acid and Scurvy (Mead Johnson)—Advanced Human Physiology Class, University of Texas, Austin.

Aureomycin, The Versatile Antibiotic (Lederle Laboratories)—Washington-Burleson Counties Medical Society, Brenham, and McMurry College, Abilene.

Back to Normal (British Information Services)—Southwestern Pre-Medical Association, Georgetown.

Be Your Age (Metropolitan Life Insurance Company)—Rotary Club, Marfa; Lions Club, Marfa; and Rotary Club, Dublin.

Breast Cancer: The Problem of Early Diagnosis (American Cancer Society)—Baylor University Pre-Medical Society, Waco, and Dr. Richard A. Lucas, Austin.

Breast Self Examination (American Cancer Society)—Brackenridge Hospital, Austin, and Stephenville Hospital and Clinic, Stephenville.

Cancer: The Problem of Early Diagnosis (American Cancer Society)—Alpha Mu Alpha, North Texas State College, Denton.

Cervical Smear (Dr. Karl J. Karnaky)—King's Daughters Clinic, Temple, and Brackenridge Hospital School of Nursing, Austin.

Cholecystectomy (Mead Johnson)—Dr. Edgar F. Jones, Jr., Galveston.

Cloud in the Sky (Texas Tuberculosis Association)—Gonzales High School, Gonzales.

Delivery of Triplets (Ciba Pharmaceutical Products)—Washington-Burlescn Counties Medical Society, Brenham, and Alpha Mu Alpha, North Texas State College, Denton.

Diagnosis of Poliomyelitis (National Foundation for Infantile Paralysis)—Drs. Rutledge and Lee, Daingerfield; Brackenridge Hospital School of Nursing, Austin; and Kimble-Mason-Menard-McCulloch Counties Medical Society, Brady.

The Doctor Speaks His Mind (American Cancer Society)—Stephenville Hospital and Clinic, Stephenville.

Feeding the Infant During the First Year (Mead Johnson)—Brackenridge Hospital School of Nursing, Austin.

Functional Anatomy of the Hand (National Foundation for Infantile Paralysis)—Wilson N. Jones Hospital Staff, Sherman.

Gastro-Intestinal Cancer: The Problem of Early Diagnosis (American Cancer Society)—Wilson N. Jones Hospital Staff, Sherman.

Goiter Surgery (Mead Johnson)—Baptist Memorial Hospital Staff, San Antonio, and Scott and White Memorial Hospitals School of Nursing, Temple.

Goodbye Mr. Germ (Texas Tuberculosis Association)—Gonzales High School, Gonzales.

The Heart: Cardiovascular Pressure Pulses and Electrocardiography (G. D. Searle and Company)—Dr. R. W. Loveless, Bastrop.

The Heart: Electrokymography, Venous Catheterization and Angiocardiography (G. D. Searle and Company)—Dr. R. W. Loveless, Bastrop.

Hepatitis, Observations on (Mead Johnson)—Albert Clinic, San Antonio.

Immunization Against Infectious Diseases (Lederle Laboratories)—University of Houston Pharmacy School, Houston.

Infantile Paralysis, Orthopedic Treatment of (Mead Johnson)—Brackenridge Hospital, Austin.

Infantile Paralysis, Your Fight Against (National Foundation for Infantile Paralysis)—Brackenridge Hospital, Austin.

Injuries, Athletic, Care and Prevention (Becton, Dickinson and Company)—Stephenville Hospital and Clinic, Stephenville.

Lesions of Vulva, Vagina and Cervix (Dr. Karl J. Karnaky)—Albert Clinic, San Antonio.

Let My People Live (Texas Tuberculosis Association)—Dr. Glenn S. Mitchell, McKinney.

Magic Bullets (U. S. Public Health Service)—Gonzales High School, Gonzales.

Malaria (British Information Services)—Brackenridge Hospital School of Nursing, Austin.

Modern Nutrition (E. R. Squibb and Company)—Dr. Glenn S. Mitchell, McKinney.

On Our Own (National Foundation for Infantile Paralysis)—Brackenridge Hospital School of Nursing, Austin, and Dr. Thomas F. Bryan, Dublin.

Once upon a Time (Metropolitan Life Insurance Company)—Dr. Thomas F. Bryan, Dublin.

Oxygen, The Administration by Oro-Pharyngeal Catheter (Mead Johnson)—McKnight Sanatorium School of Nursing, Sanatorium.

Oxygen Therapy Procedures (Linde Air Products)—McKnight Sanatorium School of Nursing, Sanatorium.

Post-Poliomyelitis Paralysis, Operative Procedures (National Foundation for Infantile Paralysis)—Methodist Hospital Surgical Staff, Dallas.

Preventive Medical Program for Children, The Significance of (Mead Johnson)—Dr. John V. Connolly, Shiner.

Proof of the Pudding (Metropolitan Life Insurance Company)—Dr. Glenn S. Mitchell, McKinney, and Rotary Club, Dublin.

Psychiatry in Action (British Information Services—Southwestern Premedical Association, Georgetown.

A Question in Time (American Cancer Society)—Junior Chamber of Commerce, Monahans.

Roentgen Pelvimetry (Mead Johnson)—Dr. Arthur J. Barnett, Monahans.

Skin Grafting of Extensive Burns (Eaton Laboratories)—Dr. Arthur J. Barnett, Monahans; Nolan - Fisher - Mitchell Counties Medical Society, Sweetwater; and Lillie Jolly School of Nursing, Houston.

Skin Studies (Procter and Gamble Company)—Dr. R. W. Loveless, Bastrop.

Sutures Since Lister (Johnson and Johnson)—12th and 27th Medical Groups, Bergstrom Air Force Base.

They Also Serve (American Medical Association)—Stephenville Hospital and Clinic, Stephenville.

Trichomonal and Monilial Vaginitis (G. D. Searle and Company) — Brackenridge Hospital School of Nursing, Austin.

Uterosalpingography, Serial (E. Fougera and Company) —Brackenridge Hospital School of Nursing, Austin.

Varicose Veins and Their Complications (Becton, Dickinson and Company) — Brackenridge Hospital School of Nursing, Austin.

Vitamin A in Human Nutrition (Mead Johnson)—Advanced Human Physiology Class, University of Texas, Austin.

Vitamins and Some Deficiency Diseases (Lederle Laboratories)—Advanced Human Physiology Class, University of Texas, Austin.

What Is Cancer? (American Cancer Society)—McKnight Sanatorium School of Nursing, Sanatorium.

BOOK NOTICES

[1]Physical Diagnosis

HARRY WALKER, M. D., F.A.C.P., *Professor of Clinical Medicine, Medical College of Virginia, Richmond. Illustrated. 461 pages. $8. St. Louis, The C. V. Mosby Company, 1952.*

In reviewing Dr. Walker's book, "Physical Diagnosis," I find the incorporated work clearly covered, with language that is easy to understand and to the point. This treatise is a worth-while addition to our Library.

Allergic Pruritus—Its Dermatologic Management

Edited by STEPHAN EPSTEIN, M. D. *Panel Discussion by* RUDOLF L. BAER, M. D., STEPHAN EPSTEIN, M. D., CARL LAYMON, M. D., FRANCIS W. LYNCH, M. D., HERBERT RATTNER, M. D., STEPHEN ROTHMAN, M. D., *and* JAMES R. WEBSTER, M. D. *Paper, 76 pages. St. Paul, Bruce Publishing Company, 1952.*

[2]Laboratory Technique in Biology and Medicine

E. V. COWDRY, *Research Professor of Anatomy and Director, Wernse Cancer Research Laboratory, Washington University, St. Louis. Third Edition. 382 pages. $4. Baltimore, The Williams and Wilkins Company, 1952.*

The third edition of this well-known text presents, in the same form utilized in previous editions, brief accounts of techniques. Standardized as well as specialized techniques are summarized and biological medical laboratory procedures are outlined and defined. Such subjects as anterior chamber transplantation, histochemical identification of antigens, Papanicolaou techniques in exfoliative cytology, and radioautography are discussed in considerable detail. The text emphasizes the importance of the constant correlation and mutual dependence of the sciences of chemistry, physics, physiology, and pathology. References are numerous, and whether one is interested in the nature of some dye, the formula for a solution, or the details of a standardized or specialized laboratory procedure, the text in its 382 pages provides an excellent source of information.

The volume is unique among laboratory texts in that the subjects are outlined in alphabetical order; hence there is no index. Also, there is no bibliography as all references are listed in their appropriate places along with the description of the subject. The third edition includes many new descriptions of technique as well as many revisions of procedures described in the previous editions. The complete address of all contributors is given so that questions may be directed to them if desired.

This new edition includes the important procedures developed in the past four years and will continue to serve as a valuable reference text in the biological and medical laboratory.

Roentgen Anatomy

DAVID STEEL, M. D., *St. John's Hospital and Evangelical Deaconess Hospital, Cleveland, Ohio. Fabrikoid, 109 pages. $8. Springfield, Ill., Charles C. Thomas, 1951.*

[3]Diabetic Glomerulosclerosis—The Specific Renal Disease of Diabetes Mellitus

HAROLD RIFKIN, M. D., F.A.C.P., *Lecturer in Medicine, College of Physicians and Surgeons, Columbia University, New York;* LOUIS LEITER, M. D., Ph. D., *Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University; and* JAMES BERKMAN, M. D., *Instructor in Pathology, College of Physicians and Surgeons, Columbia University. A Monograph in American Lectures in Metabolism. Publication 130, American Lecture Series. 102 pages. $3.50. Springfield, Ill., Charles C. Thomas, 1952.*

This monograph covers in a succinct fashion the important subject of diabetic glomerulosclerosis. The data are taken in a large degree from the personal experience of the authors. The subject matter of the book is divided into seven chapters dealing with the clinical syndrome, pathology, pathogenesis, analysis of clinical data, laboratory aids and diagnosis, differential diagnosis, and lastly, treatment.

Throughout the volume specific cases are presented and a complete clinical and pathologic history is given. The authors emphasize the importance of every physician examining carefully all of his diabetic patients so that he may not overlook this specific renal disease of diabetes mellitus. They also emphasize the frequently forgotten fact that a normal fasting blood sugar level does not rule out or confirm a diagnosis of diabetes but that a properly conducted glucose tolerance test will give the looked-for information.

The format is excellent; the printing and paper are typical of the work of the publisher; and the booklet can be unreservedly recommended to every practicing physician.

Ruptures of the Rotator Cuff

H. F. MOSELEY, M. A., D. M., *Hunterian Professor, Royal College of Surgeons of England; Assistant Professor of Surgery, McGill University. Publication 106, American Lecture Series. Fabrikoid, 90 pages. $6.50. Springfield, Ill., Charles C. Thomas, 1952.*

[4]Neurosurgery in General Practice

ADRIEN VER BRUGGHEN, M. B., Ch. M., M. S., F.A.C.S., *Clinical Professor of Neurological Surgery (Rush) University of Illinois College of Medicine; Neurosurgeon to the Presbyterian Hospital of Chicago; Formerly Attending Neurosurgeon, Cook County Hospital, Chicago. 665 pages. $14. Springfield, Ill., Charles C. Thomas, 1952.*

This book is one which the general practitioner should find helpful, for it has been written especially for him. The contents of the book are well known to the specialist in the field and the author frankly states that the book is not to be considered a textbook on neurosurgery. Dr. Ver Brugghen very aptly has used comparatively simple medical terminology without any effort to appear erudite.

In the discussion of head injuries, the principle of conservatism is obviously maintained throughout the book. The author has pointed out some of the variances in therapy

[1]*E. A. Cayo, M. D., San Antonio.*
[2]*Maynard S. Hart, M. D., El Paso.*
[3]*H. T. Engelhardt, M. D., Houston.*
[4]*Jack I. Woolf, M. D., Dallas.*

which exist between the different neurosurgical schools of thought; although some variances were not discussed, they probably are not too important in the final mortality rate in this field. All patients with severe head injuries should be under constant neurosurgical observation, for even subtle changes may indicate the need for immediate surgery. Dr. Ver Brugghen states that "the patient should be moved, if at all possible, to a neurosurgical center, in spite of the desire to have cases operated on at one's own hospital."

It is well recognized that medicine has become so complex that it is impossible for the individual physician to know the many facets of each field of specialization. Therefore, it is believed that the good general practitioner need not be required to have a comprehensive knowledge of each field; rather he should have sufficient basic knowledge to recognize when specialized help is needed and desired. With this point of view in mind, Dr. Ver Brugghen's book is recommended for the library of the general practitioner.

ORGANIZATION SECTION

AMERICAN MEDICAL ASSOCIATION

INTERIM SESSION IN DENVER

Compromise proposals on the extension of the doctor-draft law and the health care of veterans having non-service connected disabilities were adopted and a Texas general practitioner, Dr. John M. Travis of Jacksonville, was named "General Practitioner of the Year" at the interim session of the American Medical Association held December 2-5 in Denver. Total registration at the sixth annual clinical session was 6,733.

Care of veterans with non-service connected disabilities in Veterans Administration facilities proved to be the most controversial issue before the House. The matter was first brought to the House in a report by a six-man committee which urged Congress to curtail hospital services to veterans with non-service connected disabilities. The report stated that more than a half million veterans with predominantly non-service connected diseases were admitted to Veterans Administration hospitals during 1951 alone. The committee felt that hospital and medical care programs for veterans had grown into a costly federal giant with unwieldy power and contradictory authority.

After considerable debate the House adopted the report of the Reference Committee on Insurance and Medical Service to which the matter was referred, which stated in part: "... Your committee believes that the fundamental consideration of limiting the care of veterans in Veterans Administration hospitals to the two following categories is sound: '(a) Veterans with peacetime or wartime service whose disabilities or diseases are service-incurred or aggravated; and (b) Within the limits of existing facilities to veterans with wartime service suffering from tuberculosis or psychiatric or neurological disorders of non-service-connected origin, who are unable to defray the expenses of necessary hospitalization.'

"The reference committee believes that the recommendation of the special committee '... that the provision of medical care and hospitalization in Veterans Administration hospitals for the remaining groups of veterans with non-service-connected disabilities be discontinued and that the responsibility for care of such veterans revert to the individual and the community, where it rightfully belongs,' cannot be accomplished without the cooperation of Congress, veterans organizations and the medical profession. Your reference committee, after conducting exhaustive hearings, is of the opinion that the only honorable, equitable, and reasonable way to approach this problem is to recommend to the House of Delegates that the American Medical Association, the veterans organizations, the American Dental Association, the American Hospital Association, and representatives of the Department of Defense and the Veterans Administration sit down and try to reach reasonable conclusions for appropriate action from agreed upon data rather than take any precipitate action now."

The A.M.A. Council on National Emergency Medical Service recommended to the House that action on the Doctor Draft Law which expires July 1 be deferred. More specific data and satisfactory answers to important questions are needed before action can be taken, the council stated. The House directed the Board of Trustees and the Council on National Emergency Medical Service to follow developments which might affect the requirements for medical officers in the armed forces and to support legislation to provide enough medical officers to care adequately for the health needs of the uniformed armed forces. It also adopted a resolution petitioning the National Advisory Committee to Selective Service to call a meeting of all state chairmen prior to making a final recommendation to Congress with regard to reenactment of the draft measure.

Dr. Louis H. Bauer, President of the A.M.A., addressed the House of Delegates, speaking on public health facilities, voluntary health insurance plans, commercialism in medicine, and the disciplining of unethical members.

The Board of Trustees voted a $10,000 contribution to the Committee on Careers of Nursing and announced the third contribution of the A.M.A. of a half million dollars to the American Medical Education Foundation.

The House of Delegates approved the following items:

A plan advocating a federal department of health with cabinet status.

A resolution condemning use of felons in research with new drugs.

A resolution asking the Trustees to consider establishing a permanent bureau at headquarters to maintain a study of the relationship between physicians and lay ancillary specialties.

A statement by the Trustees urging the A.M.A. and state medical societies to encourage programs offering to provide the services of a physician to anyone unable to pay for them.

In addition to these actions the House of Delegates agreed with the Judicial Council that fee splitting is unethical under any guise and that physicians should render separate bills.

An estimated thirty-five million people saw one television program from the Denver meeting and fifty million saw another. More than twenty radio and television shows were produced during the four day session.

The day before the session opened a Medical Public Relations Conference was held with more than 400 persons in attendance. Dr. E. Sinks McLarty, Galveston, took part in a panel on "Building Understanding with Future M. D.'s."

RURAL HEALTH CONFERENCE

The program for the eighth annual National Conference on Rural Health February 27 and 28 at the Hotel Roanoke, Roanoke, Va., will feature nine principal speakers in its theme of "Widening the Highway to Health."

The participating speakers include Dr. F. S. Crockett, LaFayette, Ind., chairman of the American Medical Association's Council on Rural Health; Dr. Carll S. Mundy, Toledo, vice-chairman of the Council; Frank Peck, Chicago, managing director, Farm Foundation; Eugene Butler, Dallas, editor of *Progressive Farmer;* Lilyan Zindell, Perryville, Mo., administrator of Perry County Memorial Hospital; Dr. Edmund Yantes, Wilmington, Ohio; Dr. B. N. Salzman, Mountain Home, Ark.; Dr. Felix Underwood, Jackson, Miss., director, Mississippi State Department of Health; and Dr. Louis H. Bauer, Hempstead, N. Y., President, American Medical Association.

The day preceding the general session, February 26, will be devoted to an informal get-together of physicians who are responsible for rural health programs in their respective states to discuss "Doctor Participation in Community Programs."

TEXAS MEDICAL ASSOCIATION

ANNUAL SESSION LOCAL COMMITTEES NAMED

Physicians and Auxiliary members planning to attend the 1953 annual session in Houston April 28-29 are urged by officials arranging the meeting to make room reservations for their stay in Houston as soon as possible. The Shamrock Hotel, scene of convention activities, began accepting annual session reservations on January 1. As usual, requests received first will have first considerations. Persons desiring rooms should write direct to the hotel of their choice specifying the type and number of accommodations desired and mentioning their relationship to the Texas Medical Association such as committee chairman, technical exhibitor, or member of the House of Delegates. In the event that the hotel is filled, the local hotel association will provide similar arrangements at another hotel in the city. A $10 deposit should accompany requests for reservations.

Dr. Edward T. Smith, chairman of the Committee on General Arrangements, has announced the members of local committees preparing the events of the annual session in Houston. Serving with Dr. Smith on the overall committee are Drs. S. G. Ohlhausen, E. Trowbridge Wolf, W. Frank Renfrow, and William T. Arnold. Other committees are as follows:

Hotels.—J. T. Billups, chairman; C. A. Dwyer, C. Marshall Ashmore, Mavis P. Kelsey, and Kenton R. Phelps.

Information.—Ben H. Bayer, chairman; Lyman C. Blair, and B. H. Estes.

Memorial Services.—M. D. Levy, Sr., chairman; F. J. Slataper, Denton Kerr, and Claud C. Cody, Jr.

Transportation.—Bill Robins.

Golf.—J. Reese Blundell, chairman; Roy T. Goodwin, Hugh C. Welsh, and W. A. Senglemann.

Skeet.—Jack N. Bevel.

Crow Shoot.—John Roberts Phillips.

Scientific Exhibits.—Luke Able, chairman; and Karl J. Karnaky.

Technical Exhibits.—B. A. Lawrence, chairman; and S. I. Johnson.

Halls and Lanterns.—Frank M. Windrow, chairman; C. W. Yates, and J. M. Dougall.

Publicity.—W. H. Hamrick, chairman; and W. M. Wallis.

Alumni banquets will be held Monday, April 27, between 6 and 8 p. m. and arrangements are being made by the following Houston physicians: William R. Knight, III, University of Texas; John H. Barrett, Baylor University College of Medicine; Ray Morgan, Southwestern Medical School of the University of Texas; L. E. Williford, Tulane University; and Joe W. King, University of Tennessee.

Arrangements for fraternity social events, to be held Tuesday, April 28, between 6 and 7 p. m. prior to the President's banquet, are being handled by the following chairmen: H. H. Harris, Phi Chi; L. L. D. Tuttle, Phi Kappa Psi; Hampton Robinson, Phi Beta Pi; Luke W. Able, Alpha Kappa Kappa; Ray K. Daily, Alpha Epsilon Iota; and Harry Caplovitz, Phi Delta Epsilon.

Members of the Association who desire to offer scientific exhibits or motion pictures are urged to communicate with Dr. James D. Murphy, 1556 Magnolia, Fort Worth, as soon as possible. Assignment of space for technical exhibits cannot be made after February 25. Motion pictures to be shown must be approved by a reviewing committee in the central office, 1801 Lamar Boulevard, Austin, by the same date.

TELEPHONE BROADCAST DATE CHANGED

The date of the first in the 1953 series of postgraduate telephone broadcasts to be sponsored by the Council on Medical Education and Hospitals of the Texas Medical Association has been changed from February 10 to February 17. Dr. Tate Miller, Dallas, is to be moderator for this program on "Peptic Ulcer — Modern Concepts," which will originate in the Sam Thompson Room of the Texas Medical Association building in Austin beginning at 8 p. m. The change in the date of presentation is necessary to bring the February and March broadcasts within a single fiscal month so that only one bridging charge need be made for both broadcasts.

This first program for 1953 will include discussions by four physicians as follows:

Pathology of Gastric Ulcer—Dr. E. E. Muirhead, Dallas.
Modern Trends in the Medical Treatment of Ulcer—Dr. Dolph L. Curb, Houston.
Evaluation of Present Surgical Procedures for Ulcer—Dr. Asher R. McComb, San Antonio.
Benign and Malignant Gastric Ulcer—Dr. C. A. Stevenson, Temple.

A round-table discussion including some questions and answers will be held by the group with Dr. Miller presiding. Lantern slides to illustrate the topic under discussion will be sent from the Central Office to each participating society so that they may be shown during the program.

The March 10 program is "Poliomyelitis—Evaluation of Present Status" with Dr. J. M. Coleman of Austin as moderator. The remaining telephone broadcasts will be presented October 13 and November 10.

County societies which desire to participate are urged to notify N. C. Forrester, Executive Secretary of the Association, before February 1. In addition, the society should send to the Executive Secretary the name of the contact man of the county society and indicate the place at which the meeting is to be held. Complete instructions regarding costs and technical details and the change in the date of the first broadcast were sent to each county society in December.

Inquiries regarding the telephone broadcast series should be sent to the Executive Secretary, 1801 Lamar Boulevard, Austin.

NOMINATIONS FOR 1953 GP AWARD

County societies desiring to nominate physicians as General Practitioner of the Year are required to submit their nominations not later than thirty days prior to the annual session of the Texas Medical Association at which the award is to be made. Nominations for the 1953 honor should be

sent by March 28 to the Executive Secretary of the Association and to the Councilor of the candidate's district. Biographical material and other pertinent information should accompany the nomination.

From the candidates suggested by the county societies, the Board of Councilors will select two physicians. The names of these nominees will be submitted to the House of Delegates at its first meeting in Houston, and the person elected by the House to receive the award for 1953 will be given special recognition on the last day of the annual session. Winner of the honor in Texas will receive a bronze plaque and will be the state's nominee for the national award, which was presented in 1952 to a Texan, Dr. John M. Travis of Jacksonville.

PROPOSED CONSTITUTIONAL AMENDMENTS

When the House of Delegates meets at the 1953 annual session, action will be taken on several constitutional changes which were proposed at the last annual session and have been held over for action this year. Several changes have to do with membership classification, and in one instance, an amendment to the By-Laws, which ordinarily can be acted upon after the amendment has laid on the table one day, was held over pending acceptance of a related amendment to the Constitution.

Term of Vice-Speaker

At the last annual session the House of Delegates voted to add a Vice-Speaker of the House of Delegates to the list of officers of the Association. In April, 1953 the House will act on an amendment to set the Vice-Speaker's term of office which would change the second sentence of Article III, Section 2 of the Constitution to read: "The President-Elect, Vice-President, Speaker of the House of Delegates, and Vice-Speaker of the House of Delegates shall be elected for terms of one year each."

Alternate Delegates as Members of House

An amendment to make alternate delegates to the American Medical Association ex-officio members of the House of Delegates will be acted upon. This proposed change in the second sentence of Article VII, Section 1 is as follows: "The membership of the House of Delegates shall consist of (1) Delegates, elected in accordance with this Constitution and By-Laws, and ex-officio (2) the President, the President-Elect, the Vice-President, the Secretary and the Treasurer; (3) Councilors; (4) Trustees; (5) the Speaker of the House of Delegates; (6) Vice-Speaker of the House of Delegates; (7) Texas Delegates and Alternate Delegates to the A.M.A.; (8) the Chairman of the Committee on Public Relations; and (9) the members of the Council on Medical Jurisprudence and the several chairmen of the other respective councils."

Associate Membership

A proposal regarding associate membership in the Association awaits action this year. The suggested change is to amend Article II, Section 3, by the addition of a new paragraph at the end to read: "Associate membership may be granted to holders of doctor or master of science degrees from colleges or universities whose standards are equal to those required by the University of Texas. Associate membership shall only be eligible to those devoting their full time to medical education, and who shall make application to their component county medical societies and meet the other requirements necessary for regular membership. Associate members shall not be eligible to hold office or vote."

A corollary amendment was proposed for the By-Laws and was held over for action with the Constitutional change. This amendment would be to add a new Section 8 to Chapter XIII of the By-Laws to read: "Associate membership—those eligible for membership as associate members shall have a doctor's or master's of science degree and shall be devoting their entire time to medical education, and shall be assessed dues of $10 per annum. An associate member will not be eligible to hold office or vote."

The reference committee which dealt with amendments to the Constitution and By-Laws at the 1952 session, while recognizing that action would have to wait a year, recommended rewording the amendments dealing with associate membership. The proposed rewording follows:

Amend Article II, Section 3 to read: "Associate membership may be granted to holders of doctor or master of science degrees or of doctor of philosophy or master of arts degrees (or their equivalent) with major interest in science, such degrees to be from colleges or universities whose standards are equal to those required by the University of Texas. Associate membership shall be eligible to those who devote their full time to medical education and who shall make application to their component county medical societies and meet the other requirements necessary for regular membership. Associate members shall not be eligible to hold office or vote."

Substitute for the proposed amendment to Chapter XIII calling for a new Section 8, three changes as follows:

1. Amend Chapter X, Section 4 by the addition of a new paragraph at the end to read: "Associate membership may be granted to holders of doctor or master of science degrees or of doctor of philosophy or master of arts degrees (or their equivalent) with major interest in a science, such degrees to be from colleges or universities whose standards are equal to those required by the University of Texas, provided that such persons devote their full time to medical education and meet the other requirements necessary for regular membership, and provided further that such persons are not permitted to hold office or vote. Associate membership in the Texas Medical Association may be terminated by failure of the county medical society to list such members in its annual report or by failure of such members to pay dues as provided in these By-Laws."

2. Amend Chapter XIII by the addition of a new Section 6 (subsequent sections to be renumbered) to read: "Associate members shall pay dues of $10 per annum, of which $3 shall be for subscription to the TEXAS STATE JOURNAL OF MEDICINE."

3. Make necessary internal changes in the By-Laws to conform to these amendments without the necessity of submitting such changes section by section to the House for approval.

Intern and Resident Membership

An amendment to Article II, Section 1, to repeat in the Constitution the provision for intern and resident members already defined in the By-Laws also will be acted upon. The amendment is to Article II, Section 1 by the addition of a paragraph at the end to read: "Members of county medical societies who are serving internships and residencies in hospitals, as a part of their educational qualifications, and who are not in private practice, may be elected by the county societies as 'intern members' or 'resident members.' When so elected, intern or resident members shall be entitled to all of the privileges of membership in the Association, provided that they pay the annual dues as required in the By-Laws and that their names are duly reported in the annual reports of the county societies."

Military Membership

Also regarding membership is a proposed amendment to Article II, Section 1, stating: "It is also provided that county

societies may elect to military membership those members or physicians otherwise eligible and acceptable for regular membership who are at the time serving with armed forces of our country, including the Coast Guard and such similar services in time of war, such membership to terminate with their discharge from service." This amendment would permit physicians not previously members of a county society to become affiliated while in military service.

American Medical Education Foundation Chairman

Dr. S. W. Thorn, Houston, has been appointed to serve as state chairman of the American Medical Education Foundation, according to an announcement made by Dr. T. C. Terrell, Fort Worth, President of the Texas Medical Association.

COUNTY SOCIETIES

Bastrop-Lee Counties Society

December 9, 1952

(Reported by Alford R. Hazzard, Secretary)

Roy H. Morris, Jr., Elgin, was elected president of the Bastrop-Lee Counties Medical Society at its meeting December 9. Other officers include Robert W. Loveless, Bastrop, vice-president, and Alford R. Hazzard, Giddings, secretary-treasurer. The society's board of censors is composed of Robert B. Burns, Giddings; S. W. Mantzel, Giddings; J. D. Stephens, Smithville; and Chauncey G. Goddard, Bastrop. James W. Thomas, Smithville, and Sterling M. Hardt, Bastrop, are delegates.

Bell County Society

December 3, 1952

(Reported by E. R. Veirs, President)

Treatment of Pulmonary Insufficiency with Preliminary Report of the Use of Positive Expiratory Pressure—W. O. Arnold, Temple.

The following officers of Bell County Medical Society were elected at the December 3 meeting in Temple: J. Bartow Talley, president; R. A. Murray, vice-president; and H. H. Brindley, secretary. The board of censors consists of V. M. Longmire, J. W. Pittman, and F. W. Howell. The society's delegates are R. R. Curtis and A. C. Scott, Jr., while J. R. Winston and R. N. Bartels serve as alternate delegates. All are from Temple except Dr. Pittman, who is from Belton.

The sixty-four members present heard the scientific program given above and approved Jack S. Weinblatt's application for membership.

Dallas County Society

November 11, 1952

(Reported by W. W. Fowler, Secretary)

Primary Carcinoma of the Gallbladder—George G. Finney, Baltimore.

George G. Finney, assistant professor of surgery at Johns Hopkins Medical School, Baltimore, presented the annual Charles M. Rosser Lecture on the above named subject at the November 11th meeting of the Dallas County Medical Society. He was introduced by J. Warner Duckett.

Mr. Millard Heath, executive secretary, gave a short talk on medical economic news, and Elliott Mendenhall, chairman of the nominating committee, reported. There being no nominations from the floor, this report became the official ballot.

M. T. Jenkins announced that a group of interested persons in Dallas had applied to the Secretary of the State of Texas for a charter for the Dallas Poliomyelitis Institute, the purpose of which is to provide facilities for the study and care of poliomyelitis through its acute, chronic, and rehabilitative stages, and the study and care of related diseases.

Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Society

November 18, 1952

(Reported by W. P. Watkins, Secretary)

Antibiotics in Pediatrics—Milton Bessire, Abilene.
Genitourinary Problems in Children—V. A. Pate, Abilene.

The regular meeting of the Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Medical Society was held November 18 at Cisco. During the business meeting Thomas Carol Ford of Breckenridge was elected to membership in the society, and officers for 1953 were named. The new officers are Calvin W. Harris, Ranger, president; Robert L. Holmes, Breckenridge, vice-president; E. E. Addy, Cisco, secretary; P. M. Kuykendall, Ranger, delegate; J. H. Caton, Eastland, alternate delegate; and D. Ball, Cisco, censor. The scientific program was given as outlined above.

Grayson County Society

December 9, 1952

(Reported by W. D. Blassingame, Secretary)

Officers of Grayson County Medical Society for 1953 were elected at the society's regular meeting December 9 and are as follows: Vernon Tuck, Sherman, president; Paul Pierce, Denison, vice-president; W. D. Blassingame, Denison, secretary; R. W. Duncan, Denison, delegate; John Gleckler, Denison, alternate; and Frank Sporer, Van Alstyne, censor.

Stanley Clayton, Denison, and D. C. Enloe and Wilbur Carter, both of Sherman, have been appointed to serve on the committee on medical jurisprudence. Other committee appointments include Arthur Gleckler and Joseph Stout, both of Sherman, and Dr. Duncan to the committee on tuberculosis; and Max Woodward and W. I. Southerland, Sherman, and R. G. Gerard, Denison, to the committee on public relations.

Jefferson County Society

(Reported by E. Mittendorf, Executive Secretary)

H. Grady Bevil, Beaumont, has succeeded to the presidency of Jefferson County Medical Society, after having served previously as president-elect. Other officers of the society for 1953 are Dan H. Byram, Port Arthur, president-elect; C. R. Goodwin, Nederland, vice-president; Frank R. Keith, Port Arthur, secretary-treasurer; L. C. Carter, Port Arthur, delegate; J. C. Crager and L. C. Powell, Beaumont, delegates; W. Pierre Robert, Beaumont, alternate delegate; Thomas B. Matlock and Paul R. Meyer, Port Arthur, alternate delegates; Walter E. McRee, Port Arthur, censor; and C. H. Hendry and Eugene H. Lindsey, Beaumont, censors.

Lamar County Society

November 6, 1952

(Reported by J. R. Kelsey, Jr., Secretary)

Early Symptoms of Schizophrenia—Irvin M. Cohen, Galveston.

Fifteen members and seven guests of the Lamar County Medical Society heard the above named paper when they met in Paris on November 6. The group dispensed with the business meeting.

Lubbock-Crosby Counties Society

November 4, 1952

(Reported by Wallace I. Hess, Secretary)

The Lubbock-Crosby Counties Medical Society met in regular session November 4. Mr. Bill Roberts of the Salvation Army discussed the city-county welfare fund. The inadequacy of the fund was stressed and the cooperation of

the physicians of the society was invited. The discussion by several members which ensued emphasized deficiencies of the program, noting that only a small percentage of the total went toward medical care, and that the total welfare fund of $24,000 per year was only a small percentage of what communities of comparable size and location were spending on such a program.

November 25, 1952

(Reported by Wallace I. Hess, Secretary)

The called meeting November 25 of Lubbock-Crosby Counties Medical Society was attended by approximately fifty members and guests, including representatives from hospitals in Lubbock, Crosby, and Garza Counties.

Elected to offices in the society for 1953 were Wallace I. Hess, president; Sam Arnett, vice-president; Frank Goodwin, secretary; Clyde Elkins, treasurer; A. G. Barsh and Allen T. Stewart, delegates; and Byron Jenkins, alternate delegate. All officers reside in Lubbock.

Following election of officers, William H. Gordon explained the necessity for a called meeting, stating that questions had arisen regarding the society's decision to replace Red Cross Blood Bank facilities with those of Southwest Blood Bank, Inc. Upon review of the matter, society members voted to reemphasize their original decision.

Nueces County Society

December 9, 1952

(Reported by H. A. Kennedy, Secretary)

The regular monthly meeting of the Nueces County Medical Society was held in Corpus Christi December 9. At that time proposed changes in the society's constitution and by-laws were approved. Society members also approved a motion made by James Barnard to endorse the proposed school of nursing at Spohn Hospital, Corpus Christi.

Physicians accepted as new members were Euclid T. Gaddy, Doyne B. Hamilton, and Clarence J. Henry. Communications from Del Mar College, Corpus Christi, which explained a suggested course for medical secretaries, was read. The treasurer's report was accepted as read. In his presidential report, Franklin W. Yeager gave a résumé of the year's work; the new president, M. C. Kendrick, then conducted the election of other officers. Those who will serve during the coming year are H. A. Kennedy, president-elect; F. F. Rogers, vice-president; William E. Morris, secretary; L. W. O. Janssen, treasurer; C. P. Yeager and J. F. Pilcher, delegates; H. E. Griffin and Y. C. Smith, Sr., alternate delegates; and R. J. Sigler, L. C. Arnim, and F. W. Hartwick, censors. All are of Corpus Christi.

Potter County Society

November 10, 1952

(Reported by William J. Campbell, Secretary)

Industrial Accident Cases—Edward T. Smith, Houston.

Approximately fifty members attended the dinner-meeting held in Amarillo on November 10 of the Potter County Medical Society. Gaylord R. Chase, Amarillo, presided over the program, and Robert F. Hyde, Amarillo, member of the program committee, introduced the guest speaker, whose name and topic appear above. Following the lecture was presented a short film which gave supplementary information relative to the topic discussed.

During the business session, attention was called to the approaching time for payment of county and state membership dues. Robert P. Gallagher's application for membership in the society was approved. Reports were received from Emil P. Reed, chairman of the hospital emergency committee, and Thomas P. Churchill of the blood bank committee. William J. Campbell, Fred J. Crumley, and William R. Klingensmith, Jr., were appointed as a committee to secure a suitable permanent meeting place for the society. All of these members are from Amarillo.

December 8, 1952

(Reported by Mrs. Cassie Atherton, Executive Secretary)

Silent Lesions of the Lungs—Houck E. Bolton, Philadelphia.

The regular meeting of the Potter County Medical Society was held in Amarillo December 8. Following dinner Dr. Houck, assistant professor of thoracic surgery at Hahnemann Medical College and Hospital of Philadelphia, spoke on the topic given above and also presented a film on diseases of the chest. New officers of the society were elected during the business session. Fred Crumley was named president; Horace L. Wolf, vice-president; William J. Campbell, secretary-treasurer; William R. Klingensmith, junior censor; A. E. Winsett, delegate; and Don Marsalis, alternate delegate. All are of Amarillo.

The society voted to pay for a brunch to be given by the Potter County Auxiliary for teachers whose pupils will take part in the essay contest sponsored by the Association of American Physicians and Surgeons. In addition, financial reports for 1952 of both the society and its official publication, *Bulletin of Potter County Medical Society*, were read and approved.

Tarrant County Society

November 18, 1952

(Reported by W. P. Higgins, Jr., Secretary)

Indications and Value of Gastroscopy—Charles Robinson, Fort Worth.
Discussion—Kenneth Wiggins, John J. Andujar, John S. Marietta, and C. C. Garrett, Fort Worth.

Eighty-two members attended the meeting November 18 in Fort Worth of the Tarrant County Medical Society.

Kenneth L. Jorns and Irving Rapfogel were elected to membership, and the membership of Edward A. Luke was transferred from the King County (Washington) Medical Society.

An amendment regarding the society's membership dues was adopted, and the nominating committee announced candidates for the society's offices for 1953.

December 2, 1952

(Reported by W. P. Higgins, Jr., Secretary)

At a dinner meeting December 2 in Fort Worth, the following officers were named to serve Tarrant County Medical Society for 1953: William M. Crawford, president; W. P. Higgins, Jr., president-elect; Louis J. Levy, vice-president; S. W. Wilson, secretary-treasurer; John J. Andujar, board of censors; Hub Isaacks, board of trustees; J. F. McVeigh and E. P. Hall, Jr., delegates; and W. F. Armstrong and Mal Rumph, alternates.

Travis County Society

November 18, 1952

(Reported by John F. Thomas, Secretary)

Use of Water Soluble Neomycin Lotion in Treatment of Pyogenic Dermatoses—M. Allen Forbes, Jr., Austin.

The Travis County Medical Society held its monthly meeting November 18 in Austin. In addition to the scientific presentation listed above, a business session was held, at which members discussed a proposal submitted by an Austin bank to afford a means of budget payments for doctor bills.

Recent applications approved for membership in the society include those of Henry Renfert, Jr. and John R. Rainey, Jr., both of Austin. Others accepted for membership by transfer are Donald Edward Pohl from the Red River Valley Medical Society of Minnesota, John E. Douglas from the Chicago Medical Society (Cook County) of Illinois, and Alfred J. Kelly from the Harris County Medical Society, all of whom are now practicing in Austin.

December 9, 1952

(Reported by Lorraine I. Stengl, Secretary)

Without Fear (motion picture).
They Live Again (motion picture).

J. Daniel Schuhman, East Bernard, was elected president of the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society at its monthly meeting held in El Campo December 9. The society's slate of officers for 1953 includes C. V. Nichols, Richmond, vice-president; Lorraine I. Stengl, El Campo, secretary-treasurer; Leonard B. Johnson, El Campo, delegate; Harold Northington, Wharton, first alternate delegate; R. G. Johnson, New Gulf, second alternate delegate; and Ben E. Knolle, Rosenberg; R. E. Leslie, El Campo; and H. C. Matthes, Bay City, censors.

AUXILIARY SECTION

COUNTY AUXILIARIES

Bexar County Auxiliary

Mr. Paul Herder, president of the board of directors of the Children's Service Bureau, San Antonio, was guest speaker at the Bexar County Auxiliary's monthly meeting November 4 in San Antonio.

Coffee was served preceding the meeting, with Mrs. E. F. Lyon acting as chairman. Her co-hostesses included Mesdames Dean Jones, C. C. Shotts, and I. C. Skinner.

Reporting for the committee on nurse recruitment, Mrs. Phillip L. Day announced that one student has applied for a loan from the special student nurses loan fund. Mrs. Louis J. Manhoff, Jr., chairman of the committee for community service, reported that first, second, and third prize winners in the essay contest conducted by the Association of American Physicians and Surgeons and sponsored by the auxiliary on the local level will receive $50, $25, and $15, respectively. Mrs. Manhoff, reporting for Mrs. Frank M. Posey, Jr., local chairman of the contest, stated that approximately 50 per cent of the schools contacted have agreed to participate.—Mrs. L. Bonham Jones, Publicity Secretary.

Ellis County Auxiliary

The Woman's Auxiliary to the Ellis County Medical Society recently held a luncheon-meeting at which new officers were elected. Mrs. B. C. Wallace, Jr., Waxahachie, became president; Mrs. A. L. Thomas, Ennis, vice-president; and Mrs. Herbert Donnell, Waxahachie, secretary-treasurer. The auxiliary expressed appreciation to Mrs. S. H. Watson of Waxahachie for her guidance and leadership as president of the group for the past twenty-five years.

The auxiliary also voted to support the Scholarship Fund and to renew subscriptions of *Today's Health* for the local schools and library. Mrs. R. N. MacLean, Waxahachie, was welcomed as a new member.—Mrs. B. C. Wallace, Jr., President.

Officers of the Woman's Auxiliary to the Texas Medical Association: President, Mrs. Robert F. Thompson, El Paso; President-Elect, Mrs. E. W. Coyle, San Antonio; First Vice-President (Organization), Mrs. R. T. Travis, Jacksonville; Second Vice-President (Physical Examinations), Mrs. Troy A. Shafer, Harlingen; Third Vice-President (Today's Health), Mrs. P. R. Jeter, Childress; Fourth Vice-President (Program), Mrs. John D. Gleckler, Denison; Recording Secretary, Mrs. Carlos R. Hamilton, Houston; Treasurer, Mrs. Oscar Marchman, Jr., Dallas; Corresponding Secretary, Mrs. Newton F. Walker, El Paso; Publicity Secretary, Mrs. A. H. Neighbors, Sr., Austin; Parliamentarian, Mrs. Joe D. Nichols, Atlanta.

Grayson County Auxiliary

New members of the Woman's Auxiliary to the Grayson County Medical Society were honorees at a coffee given by the auxiliary on October 10 in the home of Mrs. Emmett Essin, Jr., Sherman, president. Reports by several chairmen were given, and the essay contest was discussed. It was announced that sixteen schools in Grayson County are participating in the contest.

Auxiliary members from Denison acted as hostesses for the November 7 meeting of the Grayson County Auxiliary. Glenn Maxwell, professor of philosophy at Austin College, Sherman, spoke on "Living in Today's World."—Mrs. Joseph Henry Stout, Reporter.

Nueces County Auxiliary

The annual Christmas party for members of the Nueces County Auxiliary and their husbands was held December 6 at the home of Dr. and Mrs. J. R. Riley, Corpus Christi. On the hostess committee were Mesdames W. E. Morris, Turner Moller, A. J. Ashmore, J. F. McKemie, Otis Marler, W. R. Metzger, and James Sharp. The party was attended by approximately one hundred couples.—Mrs. C. D. Stewart, Press

DEATHS

S. B. LOVELADY

Dr. Sim Bedford Lovelady, Houston, Texas, died at his home in Houston on October 8, 1952 as a result of cardiac insufficiency.

A native of Alabama, Dr. Lovelady was born at Danville, Ala., on January 11, 1909, the son of Dr. William and Ora Lee (Sherrill) Lovelady. He received his preliminary education at Morgan County High School, Hartselle, Ala.; his bachelor of arts degree in 1930 from Vanderbilt University, Nashville; and his medical degree from Tulane University School of Medicine, New Orleans, in 1934. Dr. Lovelady then served a one-year rotating internship at Charity Hospital in New Orleans. He was on the permanent staff of the Mayo Clinic, Rochester, Minn., for twelve years, including a five-year fellowship with the Mayo Foundation. During that period, Dr. Lovelady was a consulting obstetrician and gynecologist at St. Mary's Hospital, Rochester, as well as at the Clinic, and on the faculty of the Postgraduate School of the University of Minnesota Medical School, Minneapolis. He

An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

entered the Army Medical Corps in 1942 with the rank of major and served with the 182nd General Hospital in England; he was discharged with the rank of lieutenant colonel. Dr. Lovelady moved in 1950 to Houston, where he established his private practice and specialized in obstetrics and gynecology.

A member of the American Medical Association and the Texas Medical Association through the Harris County Medical Society, Dr. Lovelady also was formerly a member of Minnesota State Medical Association through Olmsted-Houston-Fillmore-Dodge Counties Medical Society. In addition, he held memberships in the Minnesota Society of Obstetrics and Gynecology and the International College of Surgeons, was a fellow of both the American College of Surgeons and the Royal Society of Medicine, and a diplomate of the American Board of Obstetrics and Gynecology. Dr. Lovelady was a member of Phi Chi medical fraternity, Sigma Nu social fraternity, the Rotary and Kiwanis Clubs, the Junior Chamber of Commerce, and the Presbyterian Church.

Dr. Lovelady was married to the former Miss Mary Thompson Greer on April 21, 1937 in Decatur, Ala. She survives him as do three sons, Hugh Greer Lovelady, William Henry Lovelady II, and Robert Lawrence Lovelady, and a daughter, Rebecca Lovelady, all of Houston; his father and mother, Dr. and Mrs. W. H. Lovelady, Hartselle, Ala.; and two sisters, Mrs. Franklin Stewart, Jr., Hartselle, Ala., and Mrs. A. B. Davidson, Montgomery, Ala.

S. R. FRANK

Dr. Susan Rosa Frank, Dallas, Texas, died October 26, 1952 at her residence in Dallas.

Born in Austin on August 25, 1893, Dr. Frank was the daughter of Abe and Sarah (Davis) Frank. After attending public schools in that city, Dr. Frank also studied at the University of Texas, Austin, from which she was graduated

DR. S. ROSA FRANK

in 1915 with a bachelor of arts degree and a teacher's certificate. She taught in the Austin public schools for several years. She obtained her doctor of medicine degree in 1922 from the University of Texas School of Medicine in Galveston. For one year Dr. Frank served an internship at Mount Sinai Hospital in Philadelphia. She also performed a portion of her residency work in Philadelphia, spending four months as a resident in contagious diseases at the Philadelphia Hospital for Contagious Diseases. In addition, Dr. Frank for eight months was a resident in pediatrics at Bellevue Hospital, New York.

From September, 1924 through June, 1927, Dr. Frank practiced in Austin. During this time she was also an instructor in anatomy at the University of Texas. Dr. Frank returned to Philadelphia in 1927 to practice and instruct. There she was associated with the Woman's Medical College of Pennsylvania and Jewish Hospital. She did work of a similar nature in Paterson, N. J., and New York. Dr. Frank resumed her practice of medicine in Texas in 1944, locating at that time in Beaumont. She moved to Dallas in 1951. Dr. Frank had been unable to practice since breaking her leg in an accidental fall in March of this year.

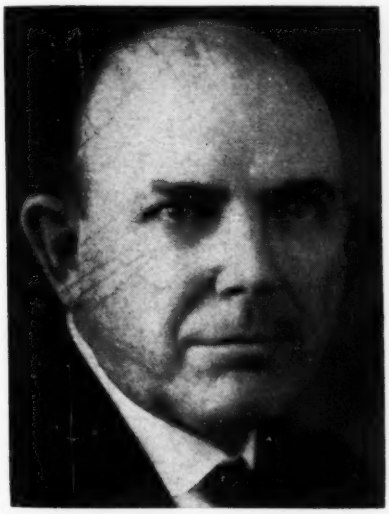
She specialized in pediatrics and was a member of the American Medical Association and the Texas Medical Association through the Travis, Jefferson, and more recently Dallas Counties Medical Societies. She was also a member of the Texas Pediatric Society.

Dr. Frank is survived by three sisters, Mrs. Arthur L. Wolf of Dallas, Mrs. Rex Logan of San Antonio, and Mrs. Waldo Forster of Austin.

W. C. KIMBROUGH

Dr. Wallace C. Kimbrough, Denton, Texas, died September 26, 1952 of cerebral hemorrhage.

Born in 1875 in Stevenson, Ala., to Mr. and Mrs. G. H. Kimbrough, Dr. Kimbrough attended William and Emma Austin Normal College in that state and was graduated in 1898 from the University of Nashville Medical Department, Nashville. He began his medical practice in Krum, Texas, and moved in 1905 to Denton where he resided until his death.



DR. WALLACE C. KIMBROUGH

Dr. Kimbrough was a member of the American Medical Association and the Texas Medical Association for many years through the Denton County Medical Society. A member of the Presbyterian Church and the Masonic Lodge, Dr. Kimbrough also had interests in farming and livestock and was a booster of the Denton County Fair, having donated land to the Denton County Fair Association a few years ago for use as fair grounds. He was honored in 1949 by the

Denton County Medical Society and the Rotary Club for his years of service to the community. The medical staff of Flow Memorial Hospital honored Dr. Kimbrough by placing his picture in the new gallery of doctors at the hospital.

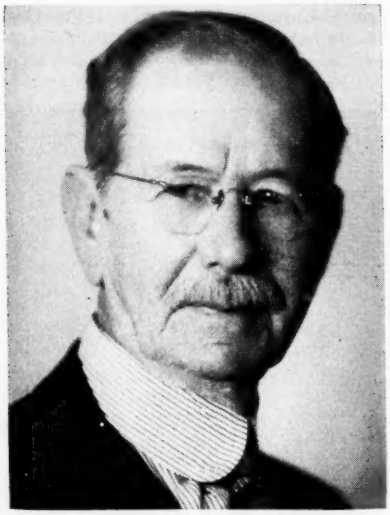
In 1899 in Denton Dr. Kimbrough married Miss Willie Smith, daughter of the late State Senator and Mrs. Emory C. Smith. Mrs. Kimbrough preceded her husband in death in 1946. A sister, Mrs. Alice Kimbrough of Dallas, survives.

G. L. DAVIDSON

Dr. Green Lafayette Davidson, Wharton, Texas, long-time member of the Texas Medical Association, died of arteriosclerotic heart disease October 31, 1952 at the home of his son, Dr. Toxey Lee Davidson of Wharton.

The son of William C. and Ann (Pruitt) Davidson, "Dr. Green," as he was known to his patients, was born March 4, 1867 at Woods Bluff, Ala. He attended public schools at Eagle Lake, and later obtained his doctor of medicine degree in 1889 from the Medical College of Alabama, Birmingham.

Dr. Davidson practiced in the vicinity of Alleyton and Eagle Lake in Colorado County until 1895, when he moved to Wharton County. Throughout the remainder of his medical career he had continued to practice in that area. Dr. Davidson was credited with having been the first doctor in Wharton County to own an x-ray machine and the first to open a hospital, the Caney Valley Hospital, in Wharton. He also helped to stamp out epidemics of influenza and malaria and worked to secure better hygiene conditions for the area.



DR. GREEN L. DAVIDSON

Dr. Davidson was a member of the American Medical Association and the Texas Medical Association through Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society. In 1947 he was elected to honorary membership in the State Association. He was a member of the Masonic Lodge for some fifty years, a senior deacon in the Baptist Church, and had served on the city council of Wharton.

In 1891 at Alleyton, Dr. Davidson married the former Miss Lulu Wright, who survives him, along with four sons, Dr. Toxey Lee Davidson, Willie Jay Davidson, and Raymond W. Davidson, all of Wharton; and Green L. Davidson, San Antonio; one daughter, Mrs. Charles A. Davis, Wharton; three brothers, Stanley C. Davidson, Baytown; Richmond S. Davidson, Ganado; and Lee Davidson, Bay City; and four sisters, Mrs. O. E. Bryan and Mrs. W. W. Lowry, both of Houston; Mrs. Herbert Bryan, Cleveland; and Mrs. B. H. Tolliver, Eagle Lake.

J. M. LEIGH

Dr. John Marshall Leigh, Midland, Texas, died November 3, 1952 in Midland, following complications from bronchial pneumonia.

The son of Henry T. and Rena (Ogilvie) Leigh, Dr. Leigh was born on March 29, 1921 at Melissa. He attended public schools in Melissa and was graduated from Thomas Jefferson High School, San Antonio. From 1939 to 1941 he was a student at San Antonio Junior College. He continued his college studies at the University of Texas in Austin and received his bachelor of arts degree in 1942. Dr. Leigh obtained his medical degree in 1945 from the University of Texas School of Medicine, Galveston.

After interning at University Hospitals, Iowa City, Iowa, Dr. Leigh first practiced in Baytown, serving as a company doctor for the Humble Oil and Refining Company. He then moved to Mason and practiced for approximately one year, before becoming associated in the general practice of medicine with his brother, Dr. Henry T. Leigh, at Midland. He continued to practice in Midland until his death. Dr. Leigh served for two years in the Navy, being stationed at Galveston and New Orleans.

DR. JOHN M. LEIGH

He was a member of the American Medical Association and the Texas Medical Association through the Harris, Kimble-Mason-Menard-McCulloch, and Ector-Midland-Martin-Howard-Andrews-Glasscock Counties Medical Societies. Dr. Leigh was also a member of Phi Chi medical fraternity, the Lions Club, Masonic Lodge, Junior Chamber of Commerce, and the Presbyterian Church, of which he was a deacon.

On July 5, 1947, Dr. Leigh married the former Miss Ethel Elizabeth Eppright. He is survived by his wife; two sons, John Marshall Leigh, Jr. and James Henry Leigh, all of Midland; his mother, Mrs. Rena Leigh, Midland; three sisters, Mrs. J. D. West, Dallas; Mrs. Lee A. Kennedy, San Antonio; and Mrs. Charles Eppright, Bay City; and a brother, Dr. Henry T. Leigh, Midland.